



Digitized by the Internet Archive
in 2018 with funding from
University of Toronto

<https://archive.org/details/medjournalApr1939uoft>

THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

(An Undergraduate Publication)

This Journal is published six times a year, November to April, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

\$1.50 per volume. 25c. per single copy.
Circulation 1,100.

ADVISORY STAFF.

Prof. E. A. Linell, M.B., Ch.B., M.D. Manc.
J. Harold Couch, M.A., M.B., F.R.C.S. Edin.,
F.R.C.S. (C.).
H. H. Hyland, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.),
D. Y. Solandt, M.A., M.D.
Prof. G. F. Marrian, D.Sc., Lond.

EDITOR-IN-CHIEF

J. H. Belton, '39.

ASSOCIATE EDITOR

K. C. Greer, B.A., '40.

MANAGING EDITOR

N. L. Hillary, '41.

ASSISTANT EDITORS

G. E. Wodehouse, '40; T. S. Wilson, '41.

WOMEN'S EDITOR

Miss D. E. Redmond, '39.

FEATURE EDITOR

J. C. McCulloch, '39.

ABSTRACTS EDITOR

D. H. Copp, '39.

CIRCULATION STAFF

Manager—D. L. Campbell, '39.

Assistants—

W. O. H. Perry, '39. C. W. Parker, '42.
E. M. J. FitzHenry, '40. J. B. Armstrong, '43.
A. J. Denne, '41. J. C. Jones, '44.

Address all communications to Room 107,
Anatomy Building.

Vol. XVI Toronto, April, 1939 No. 6

Editorial

With this issue Volume XVI is completed. The editorial staff wishes to thank all our contributors to whom any success that the Journal may have had, is due. It is the constant desire of the editors that more and more students use this Journal as a medium of

expression as only they who do, obtain full benefit from this publication.

During the past year these pages have contained many articles designed to bring to the attention of the student other phases of medical life and education than just passing examinations. Some have criticized this tendency, maintaining that the Journal should only contain articles that are of "real practical value" and which will ease the road to knowledge. Such a viewpoint is the reflection of an undeveloped mind and exemplifies the false standard of values held by many people to-day. It is easier to fill these pages with pseudo-scientific articles, tiring in their completeness, than it is to obtain the thoughtful essay that truly instructs.

The Medical Society has chosen a competent editorial board for Volume XVII. K. C. Greer as editor-in-chief, T. S. Wilson as assistant editor and N. L. Hillary as managing editor are the able members of this board who will ensure the future improvement of the Journal.

To the graduating class the Journal wishes every success in the years to come and would remind them of the words of Cowper:

Knowledge and Wisdom, far from being
one,
Have oftentimes no connection. Know-
ledge dwells
In heads replete with thoughts of other
men;
Wisdom in minds attentive to their own.
Knowledge is proud that he has learn-
ed so much,
Wisdom is humble that he knows no
more.

PRURITUS ANI — ITS CLASSIFICATION AND TREATMENT

By DR. JAMES T. DANIS, F.R.C.S. (Edin.), F.A.C.S.

Pruritus Ani may be defined as an irritative condition of the skin in the peri-anal region. Numerous causes have been suggested as the etiological factors and several classifications of the condition have been offered. The following classification appeals to me as offering some practical solution to the problem.

Group One or Symptomatic Group

In which the appearance of the peri-anal skin varies from a reddish colour to one which presents little variance from the normal. It differs from that found in the idiopathic group to be described later. The main symptoms are itching and the presence of excess moisture.

The Common *Local* causes of this variety are:—

1. Lack of cleanliness, e.g., from pockets caused by external skin tags.
2. Presence of an irritable discharge from the rectum, such as in diarrhoea, or from excess mucus resulting from prolapsing anal mucosa, or following an operation for haemorrhoids where the mucosa has been pulled down too far.
3. A pruritus secondary to
 - (a) A discharging fistula in ano and localized to or more intense in, the immediate vicinity of the fistulous opening.
 - (b) A lowly placed pilonidal sinus.
 - (c) The temporary sero-purulent discharge following operations for ischio-rectal abscess, haemorrhoids, fistula in ano, or perineal resections.
4. An acute pruritus involving the skin of the perineum and upper, inner aspects of the thighs caused by too much walking, horseback

or bicycle riding, especially during hot weather.

5. Rarely from the wearing of certain types of underwear.
6. Pin worms.

The *General* causes are those associated with infection of the urethra, prostate, vagina, or cervix; jaundice and neurosis. It is in this very limited number of cases that the peri-anal skin shows little or no change and in which itching is the only symptom.

Group Two or Mycotic Group

In which there are gross and microscopic changes in the peri-anal skin, and in which other skin manifestations are present. This group would include such conditions as epidermophyton, in which some additional evidence of the disease will be found in the skin of the groins, feet, hands or possibly the lower half of the face. The hands and feet may escape and a small ring-wormlike patch may be found in the axilla, the front of the chest, or on the abdominal wall. These require a very careful search as they may escape notice. In two of my cases the only additional manifestation was found in a small patch in the external auditory meatus and one just above the ear. The fungus may be grown on Saboraud's medium. The most acute manifestations of the disease will be found in this group; at least I have found it so in my cases. As a rule secondary infection occurs and adds to the severity of the condition. "Weeping exzema of the perineum" it was called by older writers and I think the term is very descriptive.

Group Three or Idiopathic Group

In which the peri-anal skin alone shows changes. This group makes up over 90 per cent of the cases. The peri-anal skin presents a typical appearance found in

no other condition, and once seen should never be mistaken. The first thing which impresses one is the change in colour to a uniform or patchy grey, water-soaked appearance with occasional areas of leucoplakia. The skin folds are accentuated, leading to the development of pseudo fissures. The condition may extend along the median raphe posteriorly to the tip of the coccyx and anteriorly to almost the base of the scrotum or the perineal body in the female.

Treatment

The treatment of the *FIRST* group is removal of the cause, for example; the excision of the external skin tags; ascertaining the cause of the diarrhea; the excision of prolapsing anal mucosa; the surgical treatment of a fistula. In other words, a definite cause can be found and rectified.

The treatment of the *SECOND* group belongs to the dermatologist. I may be permitted to say however, that the usual treatment adopted for "Athlete's Foot" such as Whitfield's ointment or unguentum ammoniated hydrarg. are too strong. Soothing ointments including mild analgesics and light x-ray therapy are beneficial.

The treatment of the *THIRD* group is the one I wish to deal with particularly. That this condition lends itself most stubbornly to treatment is shown by the innumerable remedies offered for its relief. In the belief that this is purely a skin lesion a countless number of ointments have been tried, with no permanent results. X-ray, ultra-violet rays, quartz light and radium have been tried by those skilled in their application but the relief has only been temporary. While no one knows the real cause of this type of pruritus ani, I am convinced that it lies in the sensory nerve fibres of this part of the skin. It would seem that any procedure which would bring about a permanent degeneration of these fibres without destroying the motor fibres would be the logical one. The operation devised by Sir Charles Ball and all other un-

dercutting operations recently described are based upon this principle, but unfortunately, after a short period of relief, regeneration takes place with the return of symptoms in about 50 per cent of cases. Subcutaneous injections of solutions in oil of various cocaine derivatives: nupercaine, procaine, and A.B.A. Solution, in the hope that their effects would be prolonged, have met with varied results. Claimed cures vary from twenty to fifty per cent. My own experience is that only a small percentage, twenty to twenty-five show a permanent cure. Any form of treatment which is not successful in at least 75 per cent of cases cannot be considered satisfactory.

For the past three and a half years, I have been using a method first introduced by Stone, and since modified by Buie. It consists of the subcutaneous injection of a 40 per cent solution of ethyl alcohol into the affected area. A long flexible needle is used, my personal preference being a Labatt needle about 4 inches in length and 20 guage. Between 25 and 35 c.c. are used. Under spinal anaesthesia the needle, fastened securely to a syringe filled with a measured quantity of alcohol, say 10 c.c., is introduced under the skin of the coccygeal region and carefully passed to a point just short of the bulbous urethra, or perineal body. As the needle is pushed forward, the skin in front of the needle is gently stretched with the fingers and thumb of the left hand sufficient to flatten out the skin folds, and at the same time the skin is "pulled" over the point of the needle. This last procedure is especially advantageous when one is curving around to the midline in front. As the needle is gradually withdrawn, the solution is evenly injected so as to iron out the skin folds. The opposite side is injected likewise, but without making a fresh puncture if possible. Complete anaesthesia is established at once.

The after treatment is particularly onerous and I know of no surgical procedure which requires more post-opera-

tive care. The injected part must be kept as clean and dry as possible. Warm saline baths are instituted the day after injection, and carried out every four hours, the skin being dried by gentle mopping and painted with an antiseptic solution such as metaphen or Scott's solution. I prefer the latter solution because its acetone content renders it more efficient as a drying agent. This procedure must be carried out for about sixteen days during which time the patient must be in the hospital. In other words, the anaesthetized portion of skin should be treated as gently as one would a skin graft. The danger period is from the tenth to the twelfth day. One of the dangers claimed for this method of treatment is sloughing and abscess formation but a little sloughing is not an undesirable feature. Careful supervision during the post-injection period will reduce the occurrence of this complication to a minimum. In my own case this complication has occurred but rarely. If an abscess forms it usually ruptures spontaneously; if not, it is opened by a puncture half an inch in length made with a scalpel, and as the skin is insensitive, no anaesthetic is required. It should be borne in mind that these abscesses are subcutaneous and not ischio-rectal in nature, hence the small opening required for efficient drainage.

I have now treated 65 cases in my Proctoscopic Clinic at St. Michael's Hospital and in 61 of these cases a permanent cure has been obtained. In one instance the treatment did no good whatsoever while in three others considerable relief was obtained. In only three patients has sloughing caused me any concern but they made a complete recovery. Small isolated areas of

sloughing have occurred in about eighteen of my cases, but this was of a minor nature and of no consequence. Most of the cases in which sloughing has occurred were associated with the development of small abscesses. In this series, one patient had had syphilis and had successfully undergone specific treatment. It bore, of course, no relationship to the onset of this disease.

Every case of pruritus ani, especially of the idiopathic variety, merits a full general examination; not that one should expect to find a definite causative factor but in order to rule out any possible contributory element. By the same token, this type of pruritus ani is never secondary to such local conditions as haemorrhoids, fissures or fistulae. However, these should be treated before attempting specific treatment of the pruritus, as a further precaution against recurrence. Occasionally one finds associated with this type of pruritus considerable redundancy of skin in the affected area and one wonders if its presence might have been the causative factor in the onset of this disease. Being a part of the body hidden from view it is impossible to obtain any help from the patient in the solution of this point. This much I am sure of; removal of the portions of excess skin and allowing the denuded areas to heal has not resulted in the disappearance of the pruritus. I have found it necessary to carry out this preliminary procedure in five of my cases; to ignore their presence and undertake specific treatment would encourage the development of sloughing. Furthermore, it is well to wait until the wounds have been completely healed for at least two weeks before proceeding with the injections.



THE INDICATIONS AND USES OF SEDATIVES IN MEDICAL PRACTICE

By ALFRED AUERBACK, M.D.

The administration of sedatives is one of the most commonly used procedures in medical practice. What sedative should be used? How much given? What is the effect? Are there any dangers involved? Will the patient develop a habit from its use? These are some of the questions the doctor must answer if he is to treat his patient intelligently.

Sedatives are analgesics which decrease the sensitivity of the central nervous system and so diminish or abolish pain. However, the close relationship of analgesics to hypnotics and anaesthetics permits discussion of these under the broad title of sedatives. Hypnotics are drugs to induce sleep while anaesthetics or narcotics depress the central nervous system abolishing certain types of sensations. Inasmuch as an analgesic may become a hypnotic when used in a larger dosage any arbitrary division of these drugs into classifications is not feasible.

Phenol Analgesic Group

These are phenol derivatives useful for reducing temperature and relief of headache, migraine, neuralgic and muscular pains, but not suitable for pain due to trauma. Aspirin (acetylsalicylic acid) is the most popular of these drugs finding widespread use for relief of headache and neuralgic pains. It is given in large doses (80 grains a day) combined with equal amounts of sodium bicarbonate in cases of rheumatic fever reducing the temperature and relieving the joint pain. Salicylic acid in these large doses upsets the stomach, consequently bicarbonate is given to counteract the acidity. Aspirin gr. x. combined with codeine gr. i. is an effective analgesic for moderately severe pain.

Phenacetin and acetanalid formerly had extensive use but little at pre-

sent. A.P.C. tablets (Pulv. Phenacetini Co. C.F.) are good analgesics where aspirin alone is insufficient.

Pyramidon (amidopyrine) has fallen into disrepute due to agranulocytosis following its use.

Bromide Group

These cause general depression of the central nervous system due to bromine ion displacing chloride ion in the brain. Small doses act on the highest centres of the brain dulling the patient mentally. In cerebral restlessness bromides are very useful. They are not analgesic and only weakly hypnotic. Their action is slow in onset and they have an enduring effect due to slow elimination. This must be remembered when using preparations such as Mist. Brom. et Chloral for a continued period especially in heart cases where there is limited fluid intake and excretion. Even in small doses poisoning may follow in a few weeks.

Alcohol and Aldehyde Group

Paraldehyde, Chloral Hydrate and ethyl alcohol are in this group although the first two are used most frequently clinically.

Paraldehyde is a safe hypnotic, very rarely habit forming, but unpleasant due to its taste and smell. Given by mouth in doses of dr. ii-iii with ice water or alcohol it is an effective hypnotic and may be used in heart disease since it has no depressant effect on the heart. In delirium tremens, thyrotoxicosis, status epilepticus, paraldehyde dr. iii in oz. iv of warm olive oil constitutes an excellent sedative for control of the patient.

Chloral hydrate is a safe efficient sedative and hypnotic. Chloral hydrate gr. xv combined with sodium bromide gr. xv forms an effective mixture (Mist. Brom. et Chloral C.F. dr. ii) and may

be given as often as 3-4 times daily in cases of cerebral restlessness. This preparation is useful in treating "neurotics," that is functional illness of the Effort Syndrome type. Nervous indigestion, palpitation and insomnia, common complaints in this type of individual may be relieved by Mist. Brom. et Chloral combined with adequate psychotherapy. It is a good hypnotic in doses of dr. ii-iii given at bed-time. Chloral Hydrate itself acts quickly so that it may be given just as the patient goes to bed and since it is rapidly excreted does not produce sleepiness the next day.

Sulphonal Group

This drug and the members of its group have little use in clinical medicine. Due to its slow, irregular absorption from the alimentary tract the time of onset of action is uncertain. Excretion of the drug is also slow and uncertain so that poisoning may ensue due to its cumulative effect if used for some time.

Barbiturate Group

These are powerful hypnotics at present enjoying widespread usage. There are a multitude of barbituric acid products but only a few will be discussed. The drugs in this series vary in their effects and duration of their actions depending on the rate of their destruction or the rate of their excretion.

Barbiturates are given by mouth but under certain conditions such as status epilepticus, eclampsia, delirium tremens, or mania especially if oral or rectal administration is resisted they may be given intramuscularly or intravenously.

Phenobarbitone (luminal) and barbitone (veronal) are slow in producing their effect but are longer-acting than other barbiturates. They have to be given at least an hour prior to bed-time and tend to produce some depression or "hangover" the next day. Due to this tendency they are very useful in cases of epilepsy especially lum-

inal which may be given in large doses depending on individual requirements. Luminal gr. $\frac{1}{4}$ t.i.d. or gr. $\frac{1}{2}$ b.i.d. is useful in management of restless patients such as in pre-operative thyroid patients. In a nervously fatigued patient luminal gr. $\frac{1}{2}$ enhances the natural tendency to sleep.

Nembutal is one of the most effective members of the barbiturate family. Its effect appears soon, producing a natural sleep of 6-8 hours, the patient awakening refreshed in the morning with no depression. The dosage varies inasmuch as gr. ss is sufficient for some patients while others require gr. i-i ss. Nembutal gr. i ss combined with codeine gr. i in the presence of pain constitutes a most satisfactory sedative.

Sodium amytal is similar in action to nembutal but more expensive and used less.

Evipal is an intravenous barbituric preparation producing a rapid effect but lasting only a short time. Consequently it finds use only as an intravenous anaesthetic for short operations.

Barbiturates are used to a large extent to suppress convulsions of central origin: tetany, eclampsia, status epilepticus, chorea, tetanus, strychnine poisoning, cocaine poisoning, and delirium tremens. If oral administration is not possible preparations such as sodium luminal gr. i-ii, sodium amytal gr. vii ss or sodium nembutal may be given intramuscularly or intravenously. In maniacal or delirious patients intravenous administration of any of these drugs will greatly facilitate control of patient.

Morphine-Opium Group

These drugs are most important in the relief of pain, depressing the nervous system and diminishing sensibility to stimuli—pain, discomfort, cough, fatigue or other disagreeable sensations. The extent of their effect depends on the dosage, a large dose being capable of producing profound depression or even coma. The members of this family most used are codeine

and morphine with heroin and opium to a minor extent.

Codeine is an excellent sedative for moderately severe pain. Given in doses of gr. ss-i ss by mouth or hypodermic it relieves distress by its analgesic action. The dosage and route of administration depends on the clinical condition of the patient. A combination of aspirin gr. x and codeine gr. i is a very satisfactory analgesic. Codeine given in the form of A.P.C. & C. tablets is good for relief of severe headache or pain. For the relief of a dry hacking cough, a preparation such as Syrup of Codeine Phosphate is excellent. In the presence of pleuritic pain codeine gr. i-i ss by hypodermic will nearly always relieve the patient.

Morphine is the strongest hypnotic and analgesic member of this group. It is used to lessen pain, to procure sleep, to suppress cough, to ease dyspnoea, to facilitate anaesthesia, and to secure muscular quiet. From this it is apparent that it may be used in a great variety of disease. However, it is essential that this drug be used with understanding and discretion. It must never be given if it interferes with the observation or diagnosis of a disease process. The constriction of the pupils it produces will interfere with the observation of a case of head injury. Similarly its administration in the presence of an acute abdomen impedes a proper diagnosis. The danger of habit formation must always be kept in mind. The possibility of side actions such as nausea, gastric upset or constipation may interfere with its use. Despite these restrictions the use of morphine and opiates in general finds numerous applications.

In severe persistent pain morphine is unequalled, the dosage depending on the clinical features of the case. It is used to dull rather than abolish sensibility, consequently small doses of gr. 1/12 repeated frequently are more satisfactory than larger doses at longer intervals. In post operative cases morphine gr. 1/6 - 1/4 may be given depending on the severity of the pain. In

the crampy distress of renal colic morphine gr. 1/4 will produce relief, reinforced by atropine which relaxes the spasm. In biliary colic morphine gr. 1/4 is given if other measures such as use of nitroglycerine or atropine to relieve the spasm are fruitless. The intense pain of coronary thrombosis requires large quantities of sedative, morphine gr. 1/4 being repeated every 20 minutes until the patient is relieved. When venesection in pulmonary oedema is inadequate morphine and atropine is given. In acute dyspnoea morphine is often used to allay the air-hunger, quiet the patient and improve the respiration by slowing the fast shallow respiratory rate. The arrest of haemorrhage anywhere in the body—haematemesis, haemoptysis, from lacerated wounds—is favoured by quieting the patient. As a pre-operative sedative morphine gr. 1/4 re-inforced by atropine gr. 1/150 and hyoscine gr. 1/200 is superior to any other medication.

Opium finds little use as a sedative. Heroin gr. 1/12 is an effective drug that finds usage only in obstetrical analgesia, in depressant cough mixtures and in cases where there is an idiosyncrasy to morphine. Pantopon gr. 1/3 may be used instead of morphine gr. 1/4 but offers no advantages warranting its use to any great extent.

Hyoscine

Hyoscine (scopolamine) is another drug finding some use as a sedative. Its effects are variable in that some individuals tend to have hallucinations and become excited. It also has some side-actions—mydriasis and excessive dryness of the mouth—which discourage its use. In acute maniacal excitement, tetanus, delirium tremens, and similar conditions hyoscine gr. 1/200 - 1/100 will control the patient. As mentioned before, the hypnotic effect of morphine is enhanced by giving hyoscine. H.M.C. tablets containing hyoscine gr. 1/100, morphine gr. 1/4 eactoid gr. 1/60 (H.M.C. 1) or hyoscine gr.

1/200 are commonly used as hypnotics for very restless patients.

General Notes On Use

The use of sedatives in medical practice entails proper understanding of the patient psychologically and clinically with administration of the proper drug to attain the desired effect. Psychotherapy is of vital importance but where more material assistance is necessary adequate sedation should be given. In a psychically upset patient reassurance by the doctor may be sufficient or may also require a placebo such as a sodium bicarbonate tablet. Sedatives should never be ordered routinely but rather from day to day or even from hour to hour as indicated by the patient's clinical condition.

In prescribing hypnotics intelligent use of these drugs is necessary. The cause of the patient's sleeplessness must be understood since it may be inability to fall asleep, it may be early awakening, or it may be due to a superficial sleep with frequent awakenings. In a nervously fatigued patient inability to fall asleep is the commonest cause of sleeplessness. Nembutal gr. ss-i ss or Mist. Brom. et Chloral dr. ii acting quickly will establish sleep, the normal rhythm continuing through the night. If sleep is only superficial or if the patient awakens early a mild hypnotic with a long duration of activity is given. The best of these are veronal (barbital) and luminal (phenobarbital) which are eminently satisfactory when given early enough for their slow onset of action to become effective by bedtime. If pain is responsible for sleeplessness an analgesic such as aspirin, phenacetin or codeine alone or combined with a hypnotic gives good results.

The frequency of administration and repetition of sedatives and hyp-

notics depends on clinical indications, on the patient's condition and on the duration of action of the drug. Thus to relieve sleeplessness only one administration may prove sufficient where as in delirious states large doses at frequent intervals may be found necessary. Medication should be discontinued as soon as possible. Never prescribe a sedative or hypnotic for a prolonged period but change to another in 2-3 weeks if continued use is still required. The possibility of habit formation and addiction must be kept in mind. The dangers inherent in narcotic administration are well-known but use of hypnotics and sedatives in general also entail this possibility. Idiosyncrasy to a drug will often explain allergic-like reactions in a patient such as asthma, urticaria or menstrual upsets or will explain changes in character and behaviour while under medication. Continued usage of a drug may produce chronic poisoning with most bizarre symptoms.

Conclusion

Sedatives play a most important role in clinical medicine. Their skilful use by combining an appreciation of the patient and his clinical condition with knowledge of the pharmacologic characteristics of the individual drug play a large part in the establishment and maintenance of a successful medical practice. Used with intelligence they constitute an invaluable aid but irrational or uncontrolled administration is fraught with great danger and may even prove disastrous to the patient. The doctor confining himself to a few drugs whose actions he knows well will obtain far better therapeutic results than one who makes use of a large number of sedative preparations without a proper understanding of their composition or action.



VENOUS THROMBOSIS AND PULMONARY EMBOLISM

By C. C. GRAY, '39.

VENOUS THROMBOSIS

Thrombosis may be defined as the formation of peculiar clots in the vessels or in the heart during life.

The thrombus is thought to begin by the deposition of a small, pearly, translucent mass of platelets, which grows by the adhesions of other platelets as they flow by. These platelets form ridges running at right angles to the flow of the stream. The platelets are welded together, so that they lose their shape and are seen in section as finely granular bands in the substance of the thrombus. The thrombus quickly gets coated with the passing leucocytes and the platelets liberate a thromboplastic substance whereby fibrinogen is converted into fibrin which hangs between the ridges of platelets entangling red cells as they pass by. At first the thrombus is predominantly red in colour although the grayish-white lamellae of platelets can be seen in a cross section. The red cells soon die and lose their hemoglobin, so that a thrombus which is chiefly red at first, becomes yellowish-gray after a time and the contrasting colour of the platelet sheets is lost in the homogeneous mass.

A thrombus occludes the vessel and the thrombus tends to be propagated distally to the next branch of the occluded vein or even beyond. However, it has been suggested that the thrombus starts simultaneously over an area up to six or eight inches in length and not at a single point in all cases as is usually thought. In speaking of this type of thrombosis we are considering those not definitely following endothelial damage or localized inflammation.

Usually the formation of thrombi is termed thrombophlebitis although in many cases there is no demonstrable inflammation nor is there any intimal damage. This means that thrombosis

may occur without previous injury to the vein and this has been termed spontaneous thrombosis. Thrombi arising from normal veins cause death more frequently from a pulmonary embolus than those associated with evident phlebitis.

Irritation of the endothelial lining of the vein from spontaneous thrombosis and from trauma causes an inflammatory repair reaction in the wall of the vein resulting usually in absorption or organization of the thrombus. As a cause of local or general signs and symptoms the phlebitis is unimportant.

Nonsuppurative thrombophlebitis of the superficial and deep veins of the leg may follow non-septic operations or develop in non-surgical cases. Another form of non-suppurative thrombophlebitis is known as phlegmasia, alba dolens or milk leg. This condition follows childbirth or a pelvic operation. It is characterized by a cramp-like pain in the calf or inner side of the thigh aggravated by movement. Marked edema rapidly develops from ankle to thigh. The thrombosed femoral vein may be palpated and is often tender. There is an extensive thrombosis of the iliac, femoral and collateral veins and this would appear to be the prime cause of the edema with obstruction to the perivascular lymphatics being a contributing factor. In this condition pulmonary emboli may occur but massive pulmonary embolism is rare. The presence of marked edema and the absence of massive pulmonary embolism differentiate this form of thrombosis and spontaneous thrombosis and support the conception of a phlebitic thrombosis of the pelvic veins as the cause.

In cases of suppurative phlebitis, pyogenic organisms cause the primary infection and the secondary throm-

bophlebitis. Detached emboli lead to the formation of small abscesses in distant organs. Cavernous sinus thrombosis may follow erysipelas, boils or carbuncles of the face in the region of the nose or upper lip and pyogenic infections of the frontal and nasal sinuses.

Thrombophlebitis migrans is a type of recurrent and migratory phlebitis with thrombosis affecting both visceral and peripheral veins. Cultures of thrombosed veins are sterile and little reaction has been found in the affected portion of the vein. The lesion suggests a spontaneous rather than a phlebitic form of thrombosis.

Etiology

About the etiology of thrombosis many theories exist and the number of theories that do exist reflect on the apparent lack of a well established cause.

Three factors are mentioned by practically all writers:—

(1) Changes in the vessel wall in the form of endothelial damage.

(2) Changes in the blood composition.

(3) Slowing of the venous return.

(1) *Changes in the Vessel Wall.*—Thrombosis may follow if the vessel wall is damaged, such that the inner and middle coats are ruptured. Also in any tissue not covered by endothelium, as a roughened and bare patch in an atheromatous aorta or an old vegetation on a heart valve. It may follow any inflammation of the vein wall. In vascular obstruction, whether due to a ligature or pressure from outside, as a tumour, a true thrombus may occur if the obstruction is incomplete but if complete, only a clot will be formed, as it was formed during life but not in a moving stream.

Other authors mention eddying of the blood stream and in support of this, thrombosis does frequently occur at the site of a valve in a vein, where the size of the lumen changes or when the lumen is encroached upon either from within or from without.

(2) *Changes in the Blood Composition.*—Physico-chemical changes in the platelets or in the blood plasma or in both would seem to be responsible for the agglutination phenomenon.

It has been shown that in the post-operative patient there is an increase in the platelets synchronous with the time when thrombosis is feared. However, this response occurs alike in old and young, whereas the incidence of thrombosis is much higher in the older age group.

Various investigations have been carried out with regard to the blood clotting factors, hoping that some one measurable change might give an indication of a tendency to thrombosis. One group found the antithrombin index to be useful in predicting a post-operative tendency to thrombosis. Recently Bancroft measured this thrombotic tendency by determinations on the plasma clotting time and the fibrinogen level. Work at the Connaught Laboratories has not, however, found this method accurate and as yet they believe there is no way of telling if there is a tendency to thrombosis.

(3) *Slowing of the Venous Return.*—Belt showed that spontaneous venous thrombosis has a high incidence in cases of actual or impending heart failure. The general conception is that this effect is to slow up the circulation. Blumgart and Weiss have shown in cases of cardiac insufficiency that the velocity of flow from peripheral veins to the right heart is definitely slower than in normal persons and that the venous pressure is increased.

The leg veins and iliac veins are the most frequent sites of venous thrombosis and this suggests further that stasis plays a part in the development of thrombosis.

With these facts in mind one can picture the train of events post-operatively, where the legs are immobile, the abdominal musculature splinted and the movement of the diaphragm restricted, thus slowing the venous return from the extremities and removing the massaging effect of the res-

piratory movements on the inferior vena cava.

Likewise a non-operative patient semi-recumbent in the gatch frame bed lies practically immobile except for movements of his hands. His breathing becomes shallow and these factors favour stagnation of the blood.

(4) *Other Factors.* (a) The relationship of obesity to thrombosis is a definite one and this may be explained either by the immobility of an obese individual or by the higher incidence of varicose veins in obese persons.

(b) Regarding the effect of trauma in the production of thrombosis there is no conclusive evidence. Thrombosis with embolism has been observed following some operations which were almost atraumatic. The site of operation appears to have little connection with the sites of thrombosis especially in those cases in which stasis is pronounced.

(c) Considerable has been written about the effects of blood pressure on thrombosis and embolism and particularly that low blood pressure was a frequent finding. That this may increase the tendency to thrombosis, by stagnation of the blood is quite likely. The degree of drop in blood pressure is more marked with spinal anaesthesia and more cases of pulmonary embolism do occur following spinal anaesthesia.

(d) In a recent series at the Toronto General Hospital 10% of cases showing pulmonary embolism had varicose veins. The same proportion of non-operative patients had varicose veins as post-operative. It is common experience that traumatic or spontaneous thrombosis of varicose veins is a frequent occurrence and often pathologists trace the origin of a pulmonary embolus to a saphenous thrombosis. Some writers recommend the bandaging of extremities showing varicose veins, while the patient is confined to bed.

Spontaneous Thrombosis

As was mentioned earlier there is often no demonstrable damage to the

endothelial lining and in these cases slowing of the stream with eddying are the prime factors in initiating thrombus formation. The question immediately arises, what is it that makes the platelets stick to the vessel wall in these cases? This might be explained by one of the various theories having to do with surface tension or electro-magnetic attraction. It has been well proven that heparin does act in such a way as to reduce the stickiness of the platelets or at any rate in preventing their adherence to the vessel lining. Would it be possible then that in these cases where thrombosis occurs that there is less heparin or heparin-like substance in the circulating blood and that in the presence of stasis the field is set for thrombosis to occur?

In spontaneous thrombosis the veins most often affected are the veins of the leg and the iliac vessels. Dehydration, anaemia and circulatory failure would appear to be predisposing factors. Often their first indication is a massive pulmonary embolus and these occur because they are not attached firmly to the vessel wall and the tendency is for large thrombi to break off.

What is the fate of a thrombus? After formation a thrombus contracts. It may then become absorbed due to two main factors. (1) Softening because of poor nourishment and (2) activity of the leucocytes in destroying the thrombus.

If absorption does not take place, then organization occurs from the spot where the endothelium was injured. Usually the thrombus shrinks away from the vessel wall and becomes endothelialized again or rarely it may become canalized with blood flowing through it.

Also some or all of the thrombus may break loose from its attachment and then become an embolus. Lime salts may be deposited in an old thrombus with the formation of a phlebolith. They are often seen in varicose veins of the leg, in the pelvic and in the prostatic veins.

PULMONARY EMBOLISM

An embolus was defined by Welch as the impaction in some part of the vascular system of any undissolved material brought there by the blood current. Routine autopsies disclose that 10% of adults showed pulmonary embolism and that more cases came from the medical ward than from the surgical in the ratio of 40 to 16.

The occlusion of a pulmonary artery only results in infarction when chronic passive congestion of the lungs is present. If infarction does occur, it is hemorrhagic and remains red. The circulation is sufficiently maintained to prevent necrosis and the infarct will disappear. This explains why old infarcts in lungs are rarely seen. An infarct is a bright red, wedge-shaped area, the base of the wedge being at the surface and covered by a thin pleural exudate which explains the clinical symptom of pleural pain and the clinical sign of a friction rub. The infarct is raised above the surrounding level and is firm to the touch. The cut surface is quite dry. The extent of the infarct may be quite small or involve a whole lobe of a lung. Microscopically the alveoli are filled with blood and their outlines are no longer visible. This explains the occurrence of bloody sputum. If the embolus is septic, there will be an abscess formed in the lung. It has been found that lung abscesses follow non-septic emboli and the etiology in this case must be similar to the formation of lung abscess following aspiration of a foreign body.

In the autopsy study usually evidence of several emboli is found—the smaller ones occurring before the large massive pulmonary embolus that causes death if two-thirds or more of the pulmonary circulation is cut off. Again it is repeated that slowing of the blood flow is probably the most important factor in predisposing to the development of the dangerous type of thrombi within the vein.

Belt places the cases of pulmonary

embolism in three well defined clinical groups:

(1) The heart failure group.

(2) This group comprises those who are cachetic or debilitated with some incurable disease.

(3) In this group are those with massive pulmonary embolus occurring in convalescent patients with a normal life expectancy.

In a large series of autopsy cases it has been shown that emboli go more frequently to the lower than the upper lobe and more frequently to the right than the left lung.

Pulmonary embolism occurs slightly more often in the male. The majority of cases fall in the age group 50-70 but pulmonary embolism does occur in young people with either surgical or medical diseases.

Signs and Symptoms

The signs and symptoms of pulmonary embolism fall broadly into two main groups:

A.—Those due to the embolism.

B.—Those due to the infarction if produced.

If the patient is a post-operative one, the embolism usually occurs from the seventh to the fourteenth day.

In the typical case of embolism of large size the patient feels a sudden pain or sense of tightness in the chest usually under the sternum. He becomes ashen in colour, anxious and then cyanotic and dyspnoeic. Pulmonary edema develops, the pulse becomes feeble or imperceptible and death follows in from a few minutes to several hours.

In lung infarction, which usually occurs twenty-four to forty-eight hours after embolism there is sudden severe pleuritic pain, dyspnoea, an irritating cough often with bloody sputum. The patient looks anxious, is cyanotic and sweats profusely. On physical examination there may be a local area of consolidation. Some infarcts cause characteristic symptoms, but no physical signs and still others neither. The phy-

sical signs of an infarct sometimes lead to the diagnosis of pneumonia.

The temperature-pulse relationship is often characteristic. After running along together suddenly there is severe pain in the chest and the pulse rapidly jumps 40-50 points. Twenty-four to forty-eight hours later the temperature gradually rises as the pulse begins to fall. The temperature and pulse then remain high for six to eight days before falling to normal.

If both trunks or the main artery are occluded, the pulmonary circulation is then completely cut off, no blood reaches the left side of the heart and cerebral anaemia ensues.

Possibly the outcome of occlusion of one of the main pulmonary branches depends on circulatory efficiency. If the condition of the circulation is normal and the heart healthy, recovery will invariably take place. If the heart is damaged, then death occurs depending on how the heart meets the added demand put on it.

Likely pulmonary embolism is more frequent than it is diagnosed and perhaps some of the post-operative and post-partum cases diagnosed as pleurisy and pneumonias are really pulmonary embolism.

Treatment of Venous Thrombosis and Pulmonary Embolism.

Here we are dealing with two problems.

(1) The treatment of the established condition.

(2) The prevention of pulmonary embolism and of venous thrombosis.

If venous thrombosis is recognized, then the treatment briefly is to keep the limb at rest, to limit the inflammation and prevent the detachment of emboli, and also elevated to assist the venous return. Heat is applied externally by means of a cradle. Heparin may be given intravenously.

Mahormer and Ochsner treat thrombophlebitis with leeches. The leeches contain hirudin, an anti-coagulant supposed to combine with thrombin. They report that if enough leeches are used,

the pain decreases, edema subsides, pulmonary embolism is less likely and the duration of the phlebitis is shortened.

In the treatment of pulmonary embolism the one essential is immobility combined with symptomatic therapy. If surgery is resorted to, then the Trendelenburg operation may be done. In cases on which this operation has been done in all centres the mortality is 93%. This figure paints a pessimistic picture of the possibilities of embolotomy for massive pulmonary emboli. Heparin given intravenously is used in cases of pulmonary embolism with the hope of preventing a second embolism.

To prevent the formation of thrombosis and embolism the patient should be encouraged to move about in bed. The legs should be elevated and massaged by the nurse if necessary.

Bancroft treats those cases with a high clotting index with sodium thio-sulphate. He gives 10 c.c. of a 10% solution intravenously for several days. At the same time he increases fluids and gives a diet low in protein and fat which will lessen the clotting tendency.

If the patient about to be operated on shows previous history of thrombosis and embolism, then the choice of anaesthetic might be considered. Spinal anaesthesia definitely lowers the blood pressure and slows the blood flow. The barbiturates as sodium pentothol increase the blood flow and elevate the temperature of the extremities and hence would lessen the fear of thrombosis.

The use of heparin has recently come into prominence and the results to date warrant its use and further investigation. Experimentally in mechanically and chemically injured vein endothelium the incidence of obstruction by thrombus was very definitely less when heparin was used than when not used.

The possibility of haemorrhage arose as it would with the increase in clotting time but heparin is rapidly eliminated and a patient whose clotting time was raised to twenty minutes

had normal clotting time in one hour after discontinuing heparin.

To date some 400 cases have received heparin post-operatively and no embolism or thrombosis recognizable has occurred.

7 cases with a history of pulmonary embolism and origin of focus as thrombophlebitis of the legs treated with heparin all improved with one exception. The one exception being a difference of opinion regarding the diagnosis.

In 28 cases of spontaneous thrombosis heparin has been used and in these no embolisms have occurred and the clinical signs and symptoms showed more rapid improvement than the control group.

In blood vessel surgery, such as embolectomy, we have the ideal place for use of heparin. Here we are mechanically injuring the vessel wall and by so doing favouring thrombus formation. Results to date show that if circulation is re-established, i.e., by feeling pulsations distal to the occlusion of the vessel, then the lumen has remained patent in those cases where heparin was used.

Heparin will not dissolve a blood clot of thrombus, so if bleeding is stopped, then heparin will not start it again. For this reason, heparin is not

given for four to twenty-four hours following operation and, if there is any question of oozing or bleeding it is not given.

Heparin is administered intravenously in sufficient quantities to keep the clotting time at fifteen minutes. The rate of flow is regulated to the individual and it is used in the proportion of 10 units of heparin to 1 c.c. of saline.

Indications for Use.

(1) Post-operative. In a patient in whom active thrombosis is detected or who has had a pulmonary embolus. But as yet there is no real way of telling who is likely to develop thrombosis.

(2) Phlebitis, embolism and blood vessel surgery, the latter of which is the ideal place for use.

(3) Blood transfusion.

(4) Coronary thrombosis and cerebral vascular thrombosis, and at the present time some work is being done on this subject.

It is, therefore, evident from this paper that the treatment and results of thrombosis and embolism are going to depend largely on being able to measure the thrombotic tendency of the individual.

MORE IMPORTANT THAN EVER BEFORE

When Dextri-Maltose was marketed in 1911 "without dosage directions on the package," Mead Johnson and Company pioneered the principle that infant feeding was a therapeutic problem. Up to that time far more babies were fed by grandmothers, neighbors, grocers, and commercial houses than by physicians. This Mead Policy was not readily accepted in the beginning, and it took many years of unceasing effort before the weight of the majority medical opinion finally led to mandatory action on the part of the Committee on Foods in 1932, whereby all makers of baby foods are now OBLIGED to omit dosage directions. The Mead Policy, however, does not stop here. It embraces other principles with which all physicians interested in the private prac-

tice of medicine are in agreement, such as (2) No descriptive circulars in packages, or in shipping cartons (for druggists to hand to patients). (3) We supply no display of Mead products for druggists' windows and counters. (4) We do not advertise Mead products to patients. (5) We give no handbills and send no letters concerning Mead products to patients. (6) We do not broadcast to the public. (7) We refer patients to physicians at every opportunity. (8) We devote a great deal of effort and resources to research and to activities that assist the private practice of medicine. Is the Mead Policy worthwhile?

MEAD JOHNSON & COMPANY,
Evansville, Indiana, U.S.A.

SOME FAMOUS MEDICAL SCHOOLS

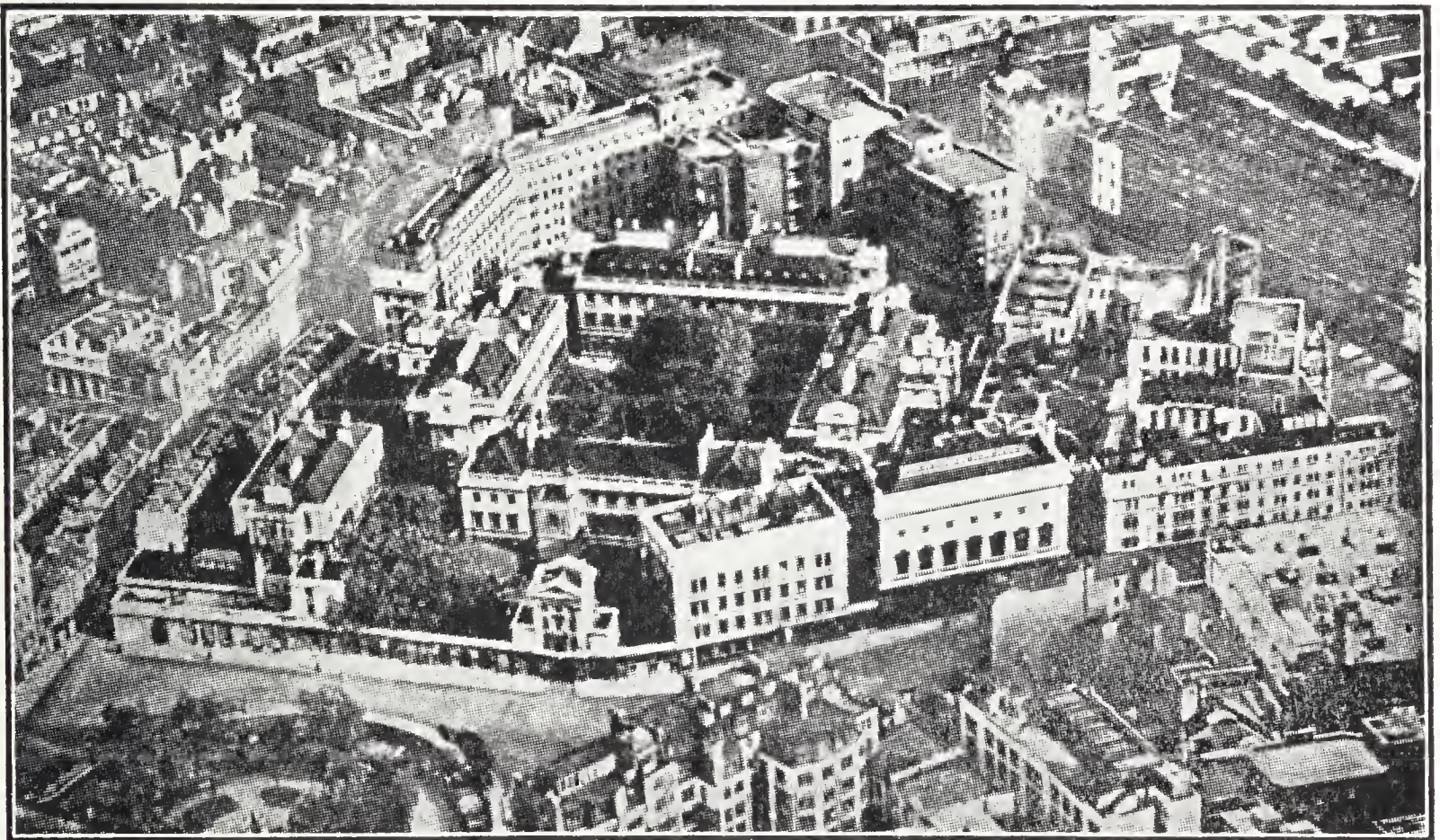
SAINT BARTHOLOMEW'S HOSPITAL, E.C. (FOUNDED IN 1123)

By CHRISTOPHER EVILL, M.B. (BARTS.)

Saint Bartholomew's Hospital is the oldest hospital in the British Empire. It was founded on its present site eight hundred and sixteen years ago.

Eight hundred and sixteen years ago the Tower of London was a new

The hospital and priory were both founded together under the name of Saint Bartholomew. The hospital was designed to give help to the needy, orphans and outcasts and to afford relief to the sick of the district. The staff



Aerial Photo Co.

Aerial View of Hospital

building and Thomas à Beckett was a boy of five. The White Ship had just gone down off Harfleur, carrying with it William, the only son of Henry I. The event is still commemorated by us because we believe that it opened the King's heart to charity and enabled Rahere, our founder, to obtain the grant of land on which the hospital is now built.

The story goes that Rahere, a canon regular of St. Austine, while on a pilgrimage to Rome, fell ill. During a dream, St. Bartholomew appeared before Rahere and commanded him to go and found a priory and a hospital on the Smooth Field (now Smithfield) in the suburbs of London. Rahere obeyed.

consisted, in those days, of a master, eight brethren and four sisters. The endowment was poor and the staff supplemented the funds by begging.

Originally a chapel stood in the centre of the site, but in 1184 Pope Lucius granted a faculty for its removal to a more suitable place, where it now stands, still within the precincts of the hospital.

In 1537, on the dissolution of the monasteries, Saint Bartholomew's Hospital came into the hands of Henry VIII, who refounded it by a royal charter. At this time the accommodation was 100 beds.

The first physician to the hospital was Roderigo Lopez, who was hanged,

drawn and quartered for compassing the death of Queen Elizabeth. Another notability of bloody fame on the staff at a later date was Dr. William Harvey, the discoverer of blood circulation.

Although the actual beginning of the medical school is not recorded, it appears that in 1662, students were in the habit of attending the medical and surgical practice of the hospital. Under encouragement from the governors the school continued to grow so that in 1726 accommodation was provided for a Museum of Anatomical and Cirurgical Preparations and leave was granted to surgeons to read lectures.

Among the early lecturers was Percival Pott and one of his pupils was John Hunter. Further additions to the course of instruction were made by John Abernethy, whose high reputation attracted a great body of students, among whom were Benjamin Brodie and the poet Shelley.

In 1843, the governors founded a Medical College, and during the latter half of the 19th century many buildings were added to it; but it continued to grow and in 1933 the college acquired the site of the Merchant Taylors' School in Charterhouse Square, which is four minutes' walk from the hospital. Here all the pre-clinical subjects are taught in new buildings, or buildings adapted for the purpose. There are squash courts and a gymnasium and other facilities for recreation. In Charterhouse Square the student spends the first two and a half years of his training.

The clinical practice of the hospital now comprises 785 beds; 269 are in the new medical block opened in 1937 and 250 are in the new surgical block opened in 1930. The cases are divided into units of 50 beds each on a floor with its own operating theatre. To each unit, or "firm," there are two members of the visiting staff, two internes and about ten students. There are medical and surgical professorial units, each with whole time professors. There are gynaecological, ENT, orthopaedic, and

septic wards, each with their own theatres.

A distinctive feature of the lay out of the hospital is the square, well known and loved by all Bart's men. It is a spacious quadrangle surrounded on three sides by blocks of wards and on the fourth by a block of administrative offices. In it are trees and a fountain, round which congregate the staff and students after lunch to smoke and chat. Across the square, members of the honorary staff hurry on their way to their rounds, collecting in their course groups of satellitic students.

The time taken between entering the hospital and taking final degrees is $5\frac{1}{2}$ years, $2\frac{1}{2}$ of which are spent in doing pre-clinical work. Many students do their pre-clinical work at Oxford or Cambridge. The three years of clinical work are divided into periods of three months. In each of these quarters, the student does a different appointment. He is entitled to two weeks' vacation each quarter. There is no long set summer vacation, as in Canadian medical schools. Another striking point of difference is the scant attention paid to public health and preventive medicine in our medical curriculum as compared with the thoroughness with which they are treated in Canadian medical schools.

The appointments of house physician or surgeon are for one year, six months resident and six months non-resident. In addition, there are resident obstetric, ophthalmic and ENT internes and three resident anaesthetists. There are numerous non-resident appointments. All are paid at the rate of \$400 per annum. All are filled by recently qualified students from the hospital.

The last ten years have seen many changes at St. Bartholomew's. It has acquired a large new medical college, new surgical and medical blocks and a one million volt X-ray plant; but in the next ten years more will be done. Many of the buildings in use are now over two hundred years old and though picturesque, are obsolete.

When one sees steel air raid shelters being delivered all over London and its parks scarred with trenches, one might query the advisability of installing all this expensive equipment—equipment vital in time of war—in the heart of the world's finest aerial target. However, the Lord

Mayor of London has launched an appeal for \$3,200,000 and the governors have decided to carry out a plan of reconstruction which, when complete, will give Saint Bartholomew's pride of place as the oldest and, at the same time, the newest hospital in the Empire.

ESSENTIAL HYPERTENSION

By R. F. GERRED, '39.

Essential Hypertension may be defined as a disease in which the blood pressure is found to be repeatedly or persistently above the normal due to increased peripheral resistance, resulting from generalized vasoconstriction of the arterioles and in which kidney efficiency is maintained or but slightly impaired. The definition immediately raises the question as to what level of pressure constitutes normality. Hunter, after a survey of one-quarter million individuals, states that the average blood pressure at age twenty-one is $\frac{\text{Systolic } 120}{\text{Diastolic } 80}$ mm.Hg.

and that an increase of 2mm. Hg. Systolic and 1 mm. Hg. Diastolic occurs for every five years beyond twenty. The normal range for systolic pressure is 15 mm. Hg. and for diastolic 8 mm. Hg. above or below the average for that age. Thus, for a man, forty years of age, the upper limit of normal pressure would be $\frac{140}{90}$ mm. Hg.

Physiology

In a previous article in the January issue of this journal it was pointed out that the raised pressure is due to a generalized increased vasoconstriction of the arterioles and not to organic disease of the vessels. It should be further pointed out that the vasoconstriction is not due to hyperactivity of the vasomotor centre and sympathetic nervous system but to an increase in the

intrinsic tone of the arterioles (Prenzmetal & Wilson, Pickering G.W.) The sympathetic nervous system continues to exert its normal vasoconstricting influence; that is to say there are two factors which are responsible for the caliber of the lumen of arterioles in Essential Hypertension:

1. Increased intrinsic tone.
2. Normal vasomotor tone.

This fact explains the poor results of such surgical treatment as sympathectomy. This operation simply withdraws the normal vasomotor tone and leaves behind the increased intrinsic tone of the arterioles. The pressure may thus be temporarily lowered in some cases but seldom to within normal limits and almost invariably the blood pressure rises again as the intrinsic tone is still further increased.

Pathogenesis

Experimental ischaemia of the kidney produced by partial constriction of the renal artery (Goldblatt technique) results in chronic hypertension in a dog which previously had, or subsequently has the other kidney removed. The disease in dogs is comparable in practically every way to the human benign form of Essential Hypertension. A severer degree of constriction of the renal artery in the dog results in a clinical and pathological picture similar to the malignant form in man. Wilson and Byrom have produced a malignant type of hypertension in rats

by partial constriction of one renal artery and leaving the other kidney perfectly intact. The lesions in the intact kidney were very similar to those in kidneys from Malignant Hypertension in humans. These animals showed no retention of N.P.N. and, accordingly, it is not necessary to postulate that kidney insufficiency is essential for the production of the Malignant form, as Goldblatt claimed.

The ischaemic kidney produces generalized vasoconstriction not by a nervous mechanism but by a humoral agent. Evidence, in support of this statement, was outlined in the previous article on this subject. At that time, it was stated a humoral agent had never been positively demonstrated. Since that article was written, Houssay, Fasciola & Taquini have published their work in which they claim to have obtained an extract from the venous blood of an ischaemic kidney in hypertensive dogs which prove to be intensely vasoconstrictive when tested on the vascular system of a certain species of toads. Blood from a normal kidney neutralized the vasoconstrictor powers of the extract. This may possibly explain the failure of others to demonstrate pressor substances as they always tested their extracts on animals with highly efficient kidneys. The work of Houssay et al has not as yet been corroborated but it appears to be sound.

The most characteristic pathological lesion found in Essential Hypertension in man is arteriolosclerosis of the kidney and, in the light of the above experimental work, it would seem probable that this lesion causes the production of a pressor substance in the kidney which is liberated into the blood stream and gives rise to generalized vasoconstriction of the arterioles.

Pathology

The symptoms and signs of Essential Hypertension are referable chiefly to the heart, brain, eye and occasionally to the kidney. Accordingly, one is not surprised that the patholog-

ical changes are most prominent in these organs.

The Heart—The heart is enlarged to a variable degree in all cases. This fact, though usually quite obvious to the eye can best be appreciated by weighing the organ. Bell and Hartzell have defined the upper limit of the weight of the normal organ as: Males 450 gm., females 400 gm. In Essential Hypertension the weight is usually 500 gm. or more.

The increase in weight is due to hypertrophy. At first the left ventricle alone is involved, and indeed, may increase to three times its normal size without hypertrophy of the other chambers. The walls (normally 15 mm. thick) become symmetrically thickened (up to 30 mm) bulge into the lumen of the right ventricle and render the left ventricle more conical in shape. The trabeculae and papillary muscles are quite massive in appearance.

When cardiac insufficiency has occurred the heart will show dilation as evidenced by enlargement of the ventricular cavity as well as hypertrophy and the external appearance of the cardiac muscle which formerly was firm, dark and glassy loses its gloss. White streaks of fibrosis with a certain degree of flabbiness become apparent.

Microscopic. All elements of the muscle cell hypertrophy, nuclei enlarge, sarcoplasm increases in amount and fibrillae are more numerous.

When dilatation supervenes there is evidence of interstitial fibrosis and muscle degeneration. There is fatty and vacuolar change in sarcoplasm, pigmentation and loss of striations of muscle fibres.

The Kidney—The external appearance of the kidney may vary somewhat. It may be slightly enlarged due to chronic passive congestion, or it may be small, firm and deeply scarred—the picture of the contracted kidney. Usually, however, the appearance closely resembles that of the normal kidney.

The capsule usually strips without difficulty and the surface is either

smooth, finely pitted or granular. The granulations correspond to areas of intact parenchyma, pittings and indentations to atrophic areas. Cross sections may reveal some thinning of cortex.

Microscopic. The majority of glomeruli are perfectly normal while a minority show all stages of progressive atrophy, wrinkling and thickening of the basement membrane of Bowman's Capsule; collapse and hyalinization of capillaries of tuft and loss of nuclei, hyalinization with fibrous encapsulation and finally complete obliteration.

The Tubules: Those tubules belonging to the atrophied glomeruli also undergo atrophy from lack of blood supply and disease.

Interstitial Tissue: With atrophy and disappearance of nephrons the fibrous tissue correspondingly increases. This process is so slow that the threshold at which kidney insufficiency develops, destruction of 65% of parenchyma is rarely reached.

The Vessels: According to Lohlein, the earliest change is a curious "lake-like" dilation of the afferent glomerular arteriole at its junction with the tuft. Then there is an eccentric deposition beneath the endothelium of the arteriole of eosinophilic hyaline material. Degeneration of the media with fatty deposition follows and reduces the lumen to a pin-point size. This process occurs in 90-98% of cases and is pathognomonic of Essential Hypertension.

The interlobular arteries show patchy fibrous placements of media which leads to bulging walls and tortuous course. Reduplication of elastic lamina and internal proliferation are also observed and these are succeeded by fatty and hyaline degeneration which narrows and finally obliterates the lumen.

The Brain: In patients who have succumbed with manifestations of hypertensive encephalopathy, autopsy most frequently reveals no focal lesions to account for the symptoms. The brain is often pale and bloodless. Occasionally oedema associated with some flattening

of the convolutions and more rarely actual softening is found.

The Retina: In the Benign form the pathological lesions are largely confined to the vessels. The arterioles show medial hypertrophy and degeneration and also some endothelial proliferation. Some pallor of the disc may be evident.

Arteriolar Lesions and Distribution

There is an obvious lack of agreement among authorities concerning the nature and distribution of the arteriolar lesions. Moritz and Oldt have made the most recent and most intensive study on this aspect of the subject. They observed three processes in arterioles in cases of Essential Hypertension:

- (1) Intimal hyalinization.
- (2) Medial hypertrophy and degeneration.
- (3) Endothelial hyperplasia.

(1) **Intimal Hyalinization:** This consists of a subendothelial accumulation of acidophilic material which appears to represent an infiltration or expansion of the ground substance between the smooth muscle of the media and endothelium. It is accompanied by swelling and disruption of the fibrils of the elastic lamina. It occurs with great frequency in the spleen, pancreas, adrenals, brain, eye and kidney in hypertensive cases but also to a certain extent in non-hypertensive cases. It is found in vessels of the Skeletal Muscle and G.I.T. in hypertensives alone.

(2) **Medial Hypertrophy and Degeneration:** Two types of hypertrophy are found which may occur together or independently; in one there is an increase in number and size of smooth muscle fibres and in the other there is an increase in intracellular collagen.

Medial degeneration with disappearance of smooth muscle cells and fibrous replacement frequently supervenes on hypertrophy. This process is rarely if ever found in normal cases, but is widespread among hypertensive cases being particularly noticeable in skeletal muscle.

(3) *Endothelial Hyperplasia*: This type of lesion consisted in a mild heaping up of endothelial cells associated with the formation of a meshwork of new elastic fibres and a marked inclination to degenerate. It was most frequently observed in the kidney.

In addition it is of interest to note that by actual measurement the caliber of the arterioles from hypertensive cases was found to be definitely smaller than in similar sized vessels from normal individuals.

From their investigation Monty and Oldt concluded that:

1. Intimal hyalinization and intimal proliferation of arterioles are the primary lesions which they believe results from an aging phenomena where as medial hypertrophy and degeneration are secondary to the increased intravascular tension.

2. Hyalinization and proliferation of the intima of renal arterioles is found in 97% of cases of Essential Hypertension and in less than 10% of non-hypertensive cases. Therefore it is the most common cause of chronic idiopathic hypertension.

Clinical Features

Incidence:

- (a) General—Fifteen percent of population over fifty years is the estimate Bell and Clawson have made.
- (b) Age—Of Onset. No definite knowledge of this has been ascertained. The Benign form seems to appear after forty but Dieho and Lutherland found that 44% of college students had a raised B.P. Of Death. 75% of deaths occur after 50 years.
- (c) Sex—Male and female suffer equally.
- (d) Type of Individual—The incidence is greater among the obese and those who live "high-tension" lives.
- (e) Nationality — Rare amongst primitive races.
- (f) Diabetics—Incidence is definite-

ly greater; 40% over forty years have hypertension.

- (g) Heredity—Heredity is a most important etiological factor. Janeway states that 50% of patients have a positive family history. O'Hare and Walker found that 68% of patients had a family history whereas 37% of normal individuals gave a similar history.

Symptoms and Physical Signs

The earliest stages of this disease are symptomless and when symptoms do appear they are vague, indefinite and transitory. Headache, frontal or occipital, usually worse in the morning, is the most common early complaint. Easy fatigability, insomnia, transient vertigo, palpitation, slight dyspnoea, digestive disturbances, frequent nose-bleeds, ringing in the ears, angenoid pains, irritability—these are the various complaints. They may or may not be sufficient to bring the patient to a doctor.

If repeatedly examined during this stage, the blood pressure will be found to be definitely raised or bordering on the upper limit of normal but to fluctuate widely so that in many instances it will approach normal. Nothing more will likely be found on physical examination even with the use of an ophthalmoscope. However, if Hines and Brown's work on the application of the cold water test for the purpose of discriminating those individuals who are liable to develop hypertension, is confirmed, then it will be possible to diagnose cases in the early stages. The above men found that if the hand is placed in water at 40 C. for two minutes, the blood pressure will rise 10 mm. Hg. in normal individuals, in hypertensive individuals or those who have a susceptibility for it, the rise in pressure is much greater, varying from 30-60 mm. Hg.

The progress of the disease is slow and many years elapse before serious symptoms become manifest. Ultimately the blood supply to the various organs

is further impaired by the superposition of organic disease upon the spastic arterioles. Disturbance of function ensues. Symptoms and signs will now be referable to the organs especially affected, notably the heart, brain and eye; rarely the kidney causes symptoms. In addition the patient will frequently begin to lose weight and the vague complaints of the early stage will become more troublesome, more constant and more severe.

Heart: Symptoms referable to the heart become more pronounced. Dyspnoea occurs on slighter exertion and becomes progressively more severe. Palpitation and anginal pains may be complained of. Examination of the heart reveals "ham-shaped" enlargement, a well-sustained heaving impulse, a booming first sound in the mitral area and an accentuated aortic second sound. The first heart sound may be replaced by a systolic murmur, due to relative insufficiency of the mitral orifice. Fluoroscopic examination confirms enlargement of the heart and may, in addition, reveal dilatation of aorta. An electrocardiogram shows left axis deviation. Both diastolic and systolic pressure are persistently raised well above the normal upper limit and the tendency to fluctuate is less marked.

Brain: The first evidence of involvement of cerebral vessels is the occurrence of headaches, insomnia, and increased irritability. Later the ability to concentrate becomes impaired, memory fails, tinnitus, vertigo and emotional instability develop. These patients suffer commonly from transient palsies and are particularly liable to have their career terminated by cerebral apoplexy.

Eye: The retina of the eye is the one site in the body where arterioles are visible and accordingly it is of the utmost importance to examine the retina in every case of hypertension. In this condition the vessels appear tortuous; the veins are obstructed where they are crossed by the arterioles and the central light reflex is increased.

Kidney: Adequacy of renal func-

tion is one of the most characteristic features of this disease. All renal function tests are normal until in the late and terminal stages. Nocturia and increased night volume are the first signs of kidney involvement according to Christian and O'Hare. A slight trace of albumin and an occasional hyaline cast are frequently found. A sudden increase in these usually indicates cardiac decompensation. Later fixation of specific gravity occurs but the point of fixation is usually definitely higher than in chronic nephritis. Blood constituents are normal throughout the course of the disease in the vast majority of patients. Less than half of the patients live long enough for any noticeable degree of insufficiency to develop.

Diagnosis

The diagnosis of Essential Hypertension in a moderately advanced stage usually presents little difficulty. According to Bell and Clawson a persistent blood pressure of $\frac{150}{90}$ or over with adequate renal function, or chronic myocardial disease with an enlarged heart, unassociated with any disease known to cause cardiac hypertrophy is Essential Hypertension.

Differentiation from arteriosclerosis is usually easy because hypertension in the latter occurs only in the so-called "episoidal crises" and even then the diastolic pressure is not raised.

Hyperthyroidism and aortic regurgitation are not readily confused with Essential because other manifestations of the disease are obvious.

Hypertension accompanying chronic nephritis may be troublesome but such cases occur in a younger age group (under 40 years) and there is marked impairment of renal function with fixation of specific gravity at a lower figure than occurs in Essential Hypertension. The other various renal diseases that cause hypertension likewise have impairment of renal function.

Coarctation of the aorta is recognized by the difference in blood pressure of the upper and lower limbs.

Tumours of the endocrine glands causing hypertension, not infrequently have other evidence of endocrine imbalance, as obesity.

Prognosis

A comparatively long life is compatible with the benign form of Essential Hypertension. The prognosis may be judged by:

1. Condition of retinal vessels.
2. Condition of myocardium and involvement of coronary vessels.
3. Adequacy of renal function.

Malignant hypertension is much more serious and accordingly prognosis must be more guarded.

In either form the terminating event is usually one of the following:

1. Myocardial failure—45%.
2. Cerebral apoplexy—20%.
3. Coronary disease—15%.
4. Uraemia—8%.
5. Miscellaneous—remaining percent.

BIBIOGRAPHY

1. Bell, E. T. & Clawson, B. J., Primary Hypertension. Arch. Path. 1928, 5.
2. Christian & O'Hara, Oxford System of Medicine.
3. Child, C.S., Path. of Experimental Hypertension. J. Exp. Med. April, 1935.
4. Fishberg, A. M., Anatomic Findings on Essential Hypertension. Arch. Int. Med. 1925.
5. Fishberg, A. M., Hypertension and Nephritis.
6. Golblatt, Harvey lecture. Bull. N.Y. Acad., Sept., 1938.

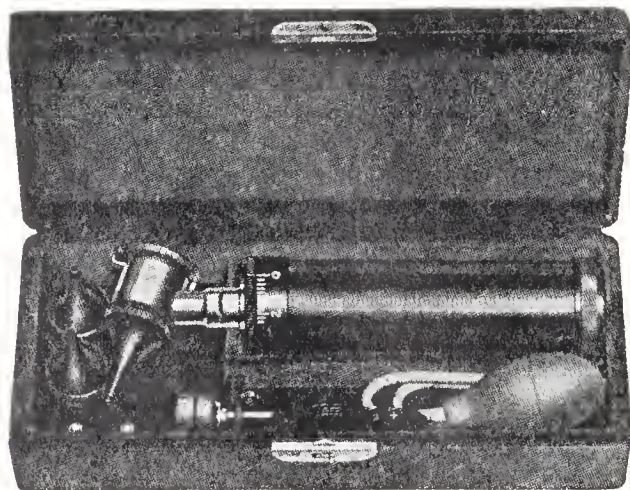
7. Haddfield & Garrod, Recent Advances in Pathology.
8. Hines & Brown. Hereditary Factor, Annals of Int. Med., 1937, 593.
9. Houssay, Fasciola & Taquini, Humoral Mech. of Essential Hyper. J. Physiol. Dec., 1939.
10. Graham, D., Arteriosclerosis and Hypertension. C.M.A.J., Jan., 1935.
11. Moritz & Oldt, Arteriolosclerosis in Hypertension, Am. J. Path., 1937, 679.
12. Pickering, G. W., Peripheral Resistance in Hypertension. Cl. Sc., 1936, 207.
13. Princmetal & Wilson, Peripheral Resistance in Hypertension. J. Clin. Invest., 1936, 63.
14. Wilson & Byrom, Experimental Malignant Hypertension. Lancet, Jan. 21, 1939.
15. Cecil. Text of Medicine.

MEDICAL MANNERS



The Simple Analogy of the Human Body and the Motor Car

By kind permission of Mr. Wm. Steig and The New Yorker.



FOR MORE PRECISE DIAGNOSIS

We invite your inspection of the

Welch-Allyn Ophthalmoscope
and Other Diagnostic Instruments

The J. F. HARTZ CO., Ltd.

PHYSICIANS' SUPPLIES

34 Grenville Street, Toronto.

1434 McGill College Avenue, Montreal, Que.

ACTINOMYCOSIS

By E. C. EVANS, '39.

Introduction.

Actinomycosis is a fungus disease which attacks cattle, horses and man. It is very common in Australia, while in Canada it is only in recent years that the so-called "lumpy jaw" of cattle has commanded much attention. This apparent increase may or may not be due to better diagnosis. From 1920-1926 but 1 case, from 1926-1931, 27 cases, and from 1931-1938, 59 cases were admitted to the Toronto General Hospital. This is one of the more common diseases which is sometimes omitted from a differential diagnosis with regrettable or even fatal results and possibly the chief value of this short discussion will be to remind you of it as an ever-present possibility.

Bacteriology.

There has been in the past much controversy regarding the transmission of this disease. Calebrook, among others, denies that it is contracted from Actinomycosis of grass or plants through ingestion or a surface injury. He suggests that it is normally present, as a saprophyte, in the mouth and intestinal tract of some people whence it invades their bodies through a break in the surface or an area of lowered vitality. Direct transmission from animals to man has in a few cases been almost definitely proven and over fifty per cent. of cases occur in people associated with farming. The actinomycosis was first observed by Bollinger in 1877 in cattle and a year later Wolff found it in man. There is a great variety of actinomycetes but in this paper we discuss only the true actinomycosis with club ends, though some believe this clinical entity may sometimes be due to forms of actinomycetes other than the actinomycosis.

There are roughly two forms of the Streptothrix Actinomycosis; the hu-

man and bovine. Both are most commonly found as small granules or colonies in pus from a lesion. These granules may be more easily obtained if a quantity of pus is taken up in a half test tube of water. The ordinary constituents of the pus are emulsified but the granules sink to the bottom and often on holding the tube up to the light may be seen as yellowish particles, hence the name sulphur granules.

The granules are colonies of the Streptothrix usually a fraction of a m.m. in diameter, soft and easily crushed under a cover slip but sometimes hard and calcified in older lesions. On the slide they are more easily seen if a drop of 20% potassium hydroxide is added. Under the microscope they appear as a rosette-like mass with an opaque dense centre. The radially arranged striations are gram positive mycelia which terminate at their outer ends in gram-negative club shaped bodies. Inside the central dense mass are seen spore-like bodies which may be degeneration products or contaminating cocci. The characteristic club ends are rarely found in cultures unless animal serum or whole blood has been used as a culture media.

Cultivation is difficult if there is much secondary infection of the pus. The common type is anaerobic but aerobic forms are occasionally found. Glucose agar or meat infusion broth are common media.

Demonstration of the Streptothrix is more difficult in pus obtained from a sinus that has been draining for some time, it is best to aspirate pus from a closed abscess if possible.

The exact relation of *B. Actinomycetum Comitans* to Actinomycosis is still unsettled, but some observers have found this in 80% of cases at some stage.

Pathology.

In general these lesions present a firm indurated mass with indefinite edges. Dense fibrosis occur and the overlying skin becomes puckered and leather-like. The mass finally breaks down and muscle, connective tissue, or bone is replaced by granulation tissue full of abscesses and draining by many sinuses. Many writers say that there is no enlargement of lymph glands while some few maintain there is a hard, painless swelling. The actinomycosis is supposedly too large to go through the lymphatic channels to the lymph glands.

The microscopic picture is that of a granuloma type of reaction plus sup-puration. Chronic inflammatory cells, fibroblasts and giant cells are seen and if the suppuration is marked the picture shows more acute inflammation. Often one may fail to demonstrate the fungus in sections but this should not mean a negative diagnosis.

Clinical Aspects.

It is convenient to discuss the clinical aspects of this disease by dividing the lesions up into groups according to their anatomical locations. The average anatomical situations of the lesions are:

Head and Neck	60%
Ileo caecal region	20%
Lungs	15%
Skin	5%

A—Head and Neck.—Roughly two-thirds of all cases occur here and most commonly in the lower jaw. The very frequent relation of this lesion to carious teeth or tooth extraction is notable. No doubt an actinomycotic infection preceded and caused many of these extractions rather than occurring after the extraction. Other times there is no previous injury or infection that is apparent and in either case the first symptom is swelling. It is later that this swelling becomes tender or painful.

The gum or outer aspect of jaw or cervical fascia develop an indurated

mass, which is quite hard. As this grows it softens, the over-lying skin becomes inflamed and more characteristically blue and mottled, and finally one or multiple sinuses form, discharging a thin watery pus. On palpation the sinuses feel very hard. Lymph gland enlargement is fairly common though many say this is due solely to secondary infection.

Sometimes the onset may resemble a boil or carbuncle which has apparently developed from a small pimple and which discharges for a few weeks before advancing to the stage described above.

More rarely the tongue is involved, a hard nodule forming beneath the surface. This extends to the surface in two to three weeks, the covering mucus membrane becomes thin, ruptures and discharges occur.

The constitutional effects are the same in a more advanced lesion regardless of its location. They are:

- (1) loss of weight.
- (2) swinging temperature 99-102.
- (3) hypochromic microcytic anaemia in late stages.

An actinomycotic lesion of head and neck is frequently confused with tuberculous adenitis or osteomyelitis of the jaw. There is usually no anaemia and the white count is about 10,000.

B—Abdomen.—The lesion here almost invariably commences in the ileo caecal region but the sigmoid colon is occasionally affected.

A mass of granulation tissue forms in the submucosa and this ulcerates through mucosa. There is great thickening of the gut wall similar to hyperplastic tuberculosis but the formation a little later of a nest of abscesses distinguishes them. Some other differences between actinomycosis and tuberculosis of caecum are: former never occurs before age of ten, latter may; former has acute and latter a more chronic course; former never affects lymph nodes and latter does; former leaves a thick puckered healing scar and latter a thin not puckered one;

former has thick viscid putrid pus and latter a thin odourless pus.

An actinomycotic lesion of ileo-caecal region occurs, generally speaking, in one of two ways: formation of an indurated mass or pseudo appendicitis. The former frequently is noticed only after patient complains of pain on extending right thigh or when the mass points to the surface above or below Poupart's ligament. The position in bed with right thigh somewhat flexed, is very characteristic.

In those cases where the first signs and symptoms resemble appendicitis the offending organ is removed and may or may not show signs of inflammation. Now one of two things may happen; the wound may continue to discharge and after three or four weeks of refusal to heal a proper diagnosis is reached, or the person may have a fairly uneventful recovery from the operation and a month or half a year later a subphrenic abscess or multiple sinuses in abdominal wall appear.

Infection of the liver nearly always occurs secondarily to an ileo-caecal lesion. It becomes so full of small abscesses as to give a honey comb appearance. There is marked increase in the fibrous septa and the liver is often adherent to adjacent structures. Of course involvement of the liver is a most hopeless complication. From a survey of abdominal cases it was found that 80% of them were fatal.

In abdominal actinomycosis there is usually a fairly well-marked anaemia and the white count is around 14,000.

C—Thorax.—Infection occurs in the lungs by aspiration of the fungus or spread from neck down or from abdomen up. Spread from lungs to pleura occurs early and at the end stage the chest wall is often full of sinuses, subcutaneous abscesses and greatly scarred. Empyema is not infrequent or there may be massive thickening of the pleura. A lower lobe is more commonly first affected and spread is often down with formation of a subphrenic abscess.

Indefinite physical signs are obtained at first, retraction, decreased fremitus and breath sounds. Chronic cough and sputum, fever, emaciation, maybe haemoptysis are some symptoms. A diagnosis of bronchiectasis, tuberculosis or carcinoma is frequently made and corrected only on finding mycelial threads in the sputum.

There is a marked anaemia, white count is 10,000 to 20,000 and the pathologist reports a fibro caseous lesion like tuberculosis with abscesses instead of cavitation. Prognosis is almost hopeless.

D—Oesophagus.—Occurs rarely, then is in upper or middle thirds and appears as an ulcer which tends to perforate resulting in mediastinitis. Diagnosis is made by biopsy.

Treatment.

Treatment may be surgical, medical, or X-ray.

Iodine therapy is an older, well tried form of treatment, although considered useless by some modern workers and is usually used as adjunct to one of the other two. Five minims of 2% tincture iodine are taken t.i.d., though some suggest far greater dosage.

Surgical treatment consists in careful excision of the sinus tracks, mere opening is not sufficient as their walls are of cartilage-like consistency. The sinuses are then packed with iodine soaked gauze.

Other people advocate injection of formaldehyde rather than excision of the sinuses. Injection of 4% formaldehyde or 1% copper sulphate along edges of the indurated area is painful but of some use. Daily injection of sinus tracts with 4% alcoholic solution of iodine is effective merely as a measure to prevent growth in the lumen of sinus tract and so prevent plugging of the tract.

A 5% alcoholic solution of thymol is said by one writer to have a specific effect. While the use of an autogenous vaccine is sponsored by many.

Radium pack and X-ray treatment

are receiving a most prominent place in treatment, and in some hospitals are used almost exclusively.

Review of Cases in T.G.H., 1931-1938.

From a review of these cases the following more striking features are listed:

- (1) usually late thirties.
- (2) usually males.
- (3) usually farmers or those associated with farming.
- (4) Of this series of 59 cases discharge notes on charts showed:
cured 7

improved	29
unimproved	12
died	11

- (5) marked frequency of history of appendectomy or tooth extraction.
- (6) Only thirteen out of fifty-nine cases in which Streptothrix could not be demonstrated in pus by smear or culture.
- (7) There have been only 11 autopsies at the Banting Institute on deaths due this cause since 1926 and these were all cases with abdominal or thoracic lesions.

TUBAL PREGNANCY — PATHOGENESIS AND SYMPTOMATOLOGY

By A. M. SUTTON, '39

Tubal pregnancy is the term used to refer to an abnormal gestation in which implantation has occurred in the Fallopian tube.

Incidence:

Schumann, who has studied this problem carefully, gives the incidence as one tubal pregnancy to every three hundred uterine implantations. A complete analysis for "tubal pregnancy" in the Toronto General Hospital was made for the period July, 1912, to February, 1925. During this period there were ten thousand and two cases of pregnancy admitted to the public wards. Of these 152 were tubal pregnancies. (The statistics quoted in the remainder of the article were obtained from the analysis of these 152 cases carried out by Dr. W. B. Hendry.)

Tubal pregnancy is naturally a disease of the child-bearing period. In this series the ages range from 19 to 41 years. The average was slightly over 29 years. Eighty per cent. of the cases occurred between 20-35 years of age.

Etiology:

No one theory of etiology fits all cases of tubal pregnancy. It is logical

to assume that it can be caused by any condition that mechanically interferes with downward passage of the impregnated ovum through the Fallopian tube. Many factors have been named as interfering with passage of the ovum. Among these are (1) tubal malformations; (2) congenital diverticula in the tubal mucosa; (3) tubal polypi and neoplasms; (4) previous pelvic inflammatory disease. This last mentioned factor is held to be the most important by the majority of writers. The frequency with which tubal pregnancy is found in tubes that have been damaged by inflammatory processes, such as result from gonorrhoea or puerperal infections, is evidence that the interruption to the ovum is the outcome of these diseases. These conditions may be manifested in the tubes by destruction of the ciliated surface epithelium or of adhesions to the external surface of the tube which prevents the normal peristalsis. If an endosalpingitis results, the most likely explanation for the interruption is that swollen or fused folds of tubal mucosa or diverticula tend to entrap the fertilized ovum during its tubal transit. The etiology may also be considered from the standpoint of the

ovum. Moll found normal ova in only ten per cent. in a series of tubal pregnancies. Whether the pathology found in the ovum is the result of improper nidation or the tubal nidation is the result of an abnormal ovum is a matter of conjecture. External wandering of the ovum, by which an ovum from one ovary, is supposed to enter the uterus by means of the opposite tube has also been mentioned as a cause of tubal implantation.

Pathology:

Except for the necessary anatomical differences, the mode of implantation is practically the same in the tubal as in the uterine pregnancy. The ovum by means of the trophoblast "eats" its way into the wall of the tube, imbedding itself there. Since there is no thick mucosa into which the ovum may imbed itself, it burrows its way into the muscle wall opening capillaries and as a result hemorrhage takes place. Should the ovum live and continue to grow, the villi invade the muscle and may actually eat through the serosa, making a small opening, through which haemorrhage may take place, into the peritoneal cavity.

Implantation may occur in either tube or in any part of one tube. In this series in 86 cases the right tube was affected and in 66 the left, while in one case there was a pregnancy in each tube. 67 were located in the ampulla, 36 in the isthmus, while three were interstitial.

Macroscopically the unruptured tubal pregnancy appears as an elastic bead-like enlargement which is dark red and congested. When rupture does not occur early there is general dilation of the tube and a marked thinning of the musculature, since the tubal musculature does not possess the power of hypertrophy inherent in the uterine musculature. Only at the site of implantation is there likely to be thickening and even in this area it may not be constant.

During the first three months of tubal pregnancy the uterus hypertro-

phies the increase being due to thickening of the endometrium and general hyperaemia and not to an increase in the musculature. A pseudodecidua vera forms which differs from that of normal pregnancy in that there is a less marked development of the spongy layer and a greater abundance of blood spaces beneath the free surface.

Terminations of Tubal Pregnancy:

A pregnancy clearly cannot continue for any considerable length of time within the narrow confines of the tube. In face of the factors which favour erosion of the tubal wall or rupture into the lumen of the tube, we find three main types of terminations:

(1) *TUBAL MOLE*: In this type of termination the trophoblast penetrates the muscle wall and opens small blood vessels to form blood sinuses and the resulting haemorrhage may be so large that the ovum is detached and dies, remaining in the tube, surrounded by a mass of coagulated blood. The dead ovum is a source of irritation, and this results in hyperaemia and often in repeated haemorrhages that may result in an haematosalpinx. Sometimes the mole is absorbed but most frequently it remains in situ. When the bleeding around the ovum is severe, some of the blood finds its way into the peritoneal cavity, from the fimbriated end of the tube and pelvic haematocoele results. The blood becomes coagulated and then encysted and adhesions form around it.

(2) *TUBAL RUPTURE*: This may be early or late. Early rupture occurs from two to five weeks and often there are no warning symptoms. In the earliest type of rupture it is common to find the ovum in the interstitial portion of the tube, and the haemorrhage may be so severe the death occurs very rapidly. Late rupture of the tube occurs from the second to the fourth month and the bleeding tends to be slower and not so profuse.

The rupture may be

- (a) intraperitoneal or
- (b) extraperitoneal.

The intraperitoneal is the usual type. The haemorrhage is commonly profuse and fatal if left alone. The opening in the tube may be very minute and the ovum usually dies. In rare cases the ovum may not be killed and may obtain a secondary attachment to the pelvic viscera and there may go to term as a secondary abdominal pregnancy.

The extraperitoneal type is rare. The rupture occurs through the under portion of the wall between the two layers of the broad ligament. In such cases the haemorrhage is not so profuse and a pelvic haematoma results. The ovum usually dies, but when this does not occur, an intraligamentary pregnancy results. This may go to term but more frequently at a later stage there is a secondary intraperitoneal rupture.

(3) *TUBAL ABORTION*: In this type the ovum is extruded through fimbriated ends of the tube and must necessarily be preceded by separation of the ovum from its attachment, so that it lies loose within the tubal lumen. The ovum is usually dead when extruded, but a secondary abdominal pregnancy may also result when the ovum is alive. The haemorrhage is usually slow and irregular but in some cases may be very profuse and even fatal. Some may take place from the uterine end of the tube but this is not usual.

In this series the tube was ruptured in 67 cases, three of which were intraligamentous. Thirty-eight showed varying stages of tubal abortions; two of which had become secondary abdominal pregnancies at $3\frac{1}{2}$ and 5 months. Twelve were unruptured and the remainder undetermined.

Symptomatology:

Before the symptomatology will be discussed a typical case history will be presented outlining the salient clinical features of the condition.

Patient, aged thirty-eight years, Para iii, was admitted to the hospital on January 14th, complaining of severe

pain over the whole of the abdomen, accompanied by intermittent vomiting and faintness. She said she had been menstruating regularly since the birth of her last baby, three years previously, and that her last period commenced on December 17th and was normal. On December 31st, she first noted some irregular bleeding which continued until January 10th, when she first consulted her doctor who gave her pills which increased the bleeding. That same evening she was seized with sharp, tearing pain in the abdomen. This pain continued for the next three days, gradually becoming less, but with occasional sudden exacerbations like a toothache. On the morning of January 14th, she awoke with severe abdominal pain accompanied by nausea, vomiting and faintness. The same afternoon she was admitted to the hospital with pulse, 95; temperature, 99° F.; haemoglobin, 58%; white blood cells, 13,800; red blood cells, 3,104,000. Examination showed no fluid blood in the abdominal cavity but marked tenderness in the left fornix, with a palpable boggy mass to the left and posteriorly.

During the administration of an enema she had a severe fainting spell and some pain but not marked. At this time there was muscular resistance over the whole lower abdomen, with dullness in the flanks. The radial pulse could not be felt and the blood pressure did not register.

Laparotomy showed the abdomen filled with blood from a left ampullary rupture, with pelvis full of clots.

Past History of Patients Having Tubal Pregnancy:

1. It has been noted that patients, in whom ectopic pregnancy is found at operation, give histories of previous pelvic inflammatory disease, intra-uterine instrumentation, laparotomy and appendicitis. A history of previous pelvic infection would account for the factors necessary to produce salpingitis, — the end result of which may cause diverticula formation and stenosis of the lumen. Appendicitis and other ex-

tra-pelvic infections may cause peritoneal adhesions with kinking of the tubes and in this way be a factor in retarding the descent of the ovum. In this series, 38 cases showed definite evidence of pelvic inflammation, 6 had had a previous ectopic, 3 cases showed ovarian disease and 1 case showed a fibroid with adhesions. In the remaining 105, there was no gross evidence of any abnormal pelvic conditions.

2. Relative sterility must also be considered as of some importance. It is unusual to find an ectopic as the 1st pregnancy without a considerable period of previous sterility. It is unusual to find an ectopic as the first pregnancy in a woman who has had a short period of co-habitation. In this series 29 had had no children, with an average period of 4-6 years sterility. 31 were Para i, 28 were Para ii, 19 were Para iii and the remainder Para iv to xiv. Sixty per cent. had a period of under four years sterility, while forty per cent. gave a history of over four years sterility. The average time elapsing from the termination of the last pregnancy was slightly under two years.

Symptoms in the History of Present Illness:

The period of gestation at which the onset of symptoms most commonly occurred was in between the fourth and eighth week, although in 22% of the cases unusual symptoms were noted before the fourth week and in 20% after the eighth week.

(1) *AMENORRHOEA*: Tubal pregnancy in its earliest stages (i.e. before any haemorrhage takes place) shows all the symptomatology of a normal intra-uterine pregnancy. In many cases the patient will first notice a missed period. However, a certain percentage of ectopics result in intra-abdominal haemorrhage without a definite history of a missed period. Frequently, at about the time of the expected period there is some irregular bleeding which is mistaken by the patient for menstruation. In this series, 80, had missed one

period; 21, had missed two; 9, had missed three; 1, had missed 5; 1, had missed 6. 34 have no history of amenorrhoea. Thus a history of amenorrhoea was obtained in about 80% of cases.

(2) *IRREGULAR VAGINAL BLEEDING*: A common symptom before rupture is irregular bleeding. It is usually but not always slight in amount and darker in colour than normal menstrual bleeding. This haemorrhage is taken as an indication of a threatened interruption of the pregnancy. The amount of the haemorrhage, which is usually small in proportion to the pain, is significant when one wishes to differentiate between threatened intra-uterine abortion and ectopic gestation. There was no bleeding in 20% of the present series and profuse bleeding in 6.5% while in the remaining 73.5% the bleeding was slight. The bleeding has been described as due to effort of the uterus, working in sympathy with the tube to repel a foreign body which in the case of the uterus is the decidua vera. Blood in the proximal end of the tube manifestly coming from the bed of a partially separated ovum situated in the isthmus, or the interstitial portion would lead one to the conclusion that some of this blood must find its way out through the uterus and that in case of a tubal abortion, the external haemorrhage is a mixture of tubal and uterine blood. A more recent theory suggests that when the ovum is separated from the tubal wall by haemorrhage, the inhibiting action of the corpus luteum ceases to act. This loss of inhibition allows the uterine bleeding to occur.

(3) *PAIN*: There is practically always abdominal pain. It may be any degree or kind and may have almost any location in the pelvis or lower abdomen. The pain has been described as sudden, severe, stabbing, cramp-like, gnawing, dull, diffuse or of indefinite character. It may be localized toward the side of the affected tube or diffuse being over the whole abdomen, in the epigastrium, below the costal margin,

in the lumbar region or radiating down the thigh. Pain may occur on defaecation and urination or on motion of the cervix. This pain is due to peritoneal irritation, as a result of a foreign material in the peritoneal cavity. In a certain percentage of cases there was radiation of the pain to the shoulder caused by subdiaphragmatic irritation due to free blood in the peritoneal cavity.

When the pain is sudden in onset and severe, it is usually accompanied by nausea and vomiting, together with fainting and collapse, usually signifying a profuse haemorrhage into the peritoneum.

The cause of the early pain may be due to (1) sudden distention of the tube due to haemorrhage (yet there is no pain when an occluded tube is distended with CO₂ in tubal inflation); (2) hyperperistalsis of the muscle of the tube in the attempt to get rid of the parasitic ovum; (3) peritoneal irritation caused by the expulsion of blood from the fimbriated end of the tube into the peritoneum.

In 33 cases the pain preceded and in 38 cases followed the haemorrhage, while in 44 cases both pain and haemorrhage occurred simultaneously. Pain was absent in only 7 cases.

(4) *DECIDUAL CAST*: The fact that the ovum is lodged in the tube does not prevent the uterus from preparing itself for the imbedding of the impregnated ovum. The endometrium, due to the stimulation of the ovaries, undergoes decidual changes. Should the ovum die, the corpus luteum ceases to function and with this loss of function there is cessation of stimulation of the endometrium. When this stimulation ceases, the decidua is cast off and is discharged either as a cast in one piece or in several small pieces.

(5) *SYNCOPE AND COLLAPSE*: Unless there has been some intraperitoneal haemorrhage, fainting or syncope are probably never noted. When blood is slowly exuded from the fimbriated end of the tube, the patient may feel faint, and will go into shock

if the bleeding is long continued. However, it is when there is rupture of the tube, with sudden intraperitoneal haemorrhage, that they go suddenly into shock. The haemorrhage in itself may not be of sufficient severity to cause collapse, but the sudden expulsion of a quantity of blood into the peritoneal cavity causes a marked irritation of the peritoneum with associated shock.

(6) *ANAEMIA*: This depends to a great extent on the pathology and type of ectopic present. If there is a profuse haemorrhage into the peritoneal cavity, the patient is usually pale and after a time has a lowered blood count. In the unruptured type with a small amount of blood trickling from the fimbriated end of the tube, an anaemia of any extent is usually not present. Patients who have an old ectopic with a continuous small amount of haemorrhage from the fimbriated end of the tube into the peritoneal cavity of 1-2 months duration associated with uterine bleeding, show a rather severe anaemia.

(7) *NAUSEA, VOMITING, ABDOMINAL DISTENTION*: The nausea and vomiting are frequent symptoms and may be due to the pregnancy per se or due to peritoneal irritation. Abdominal distention is also frequently seen. A moderate ileus develops reflexly as result of blood in the peritoneal cavity. When a patient is seen with an anomalous menstrual period and a moderate ileus with distention and ectopic pregnancy should be seriously considered.

(8) *PULSE*: This depends upon the amount of blood the patient has lost. If she has lost very little blood, there may be little change in the pulse rate. Sudden severe haemorrhage plus shock causes a rapid thready pulse.

(9) *TEMPERATURE*: As a rule the temperature is low as blood in the peritoneal cavity does not cause any marked temperature reaction. However, in old ectopics when the localized blood masses become infected, the temperature may rise markedly. On admission 54 had a temperature below 99° F.,

64 between 99-100° F., 21 between 100-101° F., and 13 above 101° F.

(10) *LEUCOCYTOSIS*: Blood in a serous cavity always causes leucocytosis. A leucocyte count was made in 110 cases. No count was under 7,000, 34 were between 7-10,000; 20 between 10-15,000; 32 between 15-20,000 and 30 over 20,000—the highest count being 41,000. The degree of leucocytosis varied with the amount and freshness of the blood in the peritoneal cavity. A white count of from 7-12,000 was found when the pelvis contained old clots formed as a result of slow leakage from the tube and the more the leakage, the higher was the count. A leucocytosis over 15,000 accompanied fresh blood in the peritoneal cavity. A differential count was done in a small number of cases and showed no marked increase in the percentage of polymorphs with no perceptible change in the percentage of eosinophils.

(11) *MASSES IN THE PELVIS*: In a ruptured ectopic, one may feel absolutely nothing in the patient's pelvis. If she has a large haematocoele one may obtain a sensation of bogginess in the fornices due to blood. Occasionally clotted blood gives the sensation of a mass. In haemorrhage from the fimbriated end of the tube, a mass may develop, due to peritoneal adhesions forming around the clotted blood. When there is a slow leak for a long time, dense adhesions form and as a result of continued small haemorrhages, the mass grows larger and harder.

(12) *BLOOD PRESSURE*: In the tubal abortion (when there is as a rule no rupture with shock and haemorrhage) there is very little change in blood pressure. In tubal rupture cases there is sudden drop in blood pressure, due to shock and haemorrhage. The blood pressure may go down to zero. If the blood pressure of such a case is taken every half hour one will find it to gradually rise until it reaches 90 or 100 and then another haemorrhage takes place, and the pressure drops again. The first drop in pressure is due to shock caused

by the sudden passage of a large amount of blood into the peritoneal cavity. Owing to the decrease in pressure, the haemorrhage from the tube ceases and a blood clot forms in the rupture opening. When the bleeding stops, the blood pressure rises again and may repeat the process.

(13) *SEDIMENTATION TIME*: This is usually longer than thirty minutes. If the ectopic is an infected one, a more rapid sedimentation time may be expected. The reason for the fairly rapid sedimentation time is probably haemorrhage.

Summary:

(1) Tubal gestation may occur in any stage of sexual life. However, it is more common at and before the age of 30 than after that age.

(2) Many factors may lead to the arrest of the impregnated ovum in the Fallopian tube, but the most important is a pre-existing chronic salpingitis resulting in a condition, in which the folds of the mucous membrane become stuck together, deep glandular pockets formed and the cilia lost, all contributing to entrap the ovum as it passes through the tube.

(3) The sequelae of tubal nidation are (1) tubal mole; (2) tubal rupture; (3) tubal abortion; (4) haematosalpinx; (5) pelvic haematocoele; (6) secondary abdominal pregnancy; (7) intraligamentous pregnancy; (8) abdominal lithopaedon.

(4) Relative sterility must be considered as a factor of significance in tubal pregnancy.

(5) The period of gestation at which the onset of the symptoms most commonly occur is between the fourth and eighth week.

(6) The symptomatology may be divided into two main groups:

(a) Those associated with pregnancy:

- (1) Amenorrhoea.
- (2) Irregular vaginal bleeding.
- (3) The decidual cast.
- (4) Nausea and vomiting.

(b) Those associated with internal haemorrhage and resultant peritoneal irritation:

- (1) Pain; abdominal, pelvis, shoulder.
- (2) Syncope; collapse.
- (3) Anaemia.
- (4) Abdominal distension.
- (5) Masses in the pelvis.
- (6) Changes in pulse, temperature, blood pressure, white blood cell count and sedimentation rate.

BIBLIOGRAPHY.

- (1) Boyd—Surgical Pathology.
- (2) Falk-Ectopic Pregnancy—American Jour. of Surgery—Vol. 37, 1937.
- (3) Frank—Obstetrical and Gynaecological Pathology.
- (4) Hendry, W. B.—Gynecological Transactions—Vol. 50, 1925—A Clinical Analysis of 152 Cases of Ectopic Gestation—P. 186.
- (5) Miller—An Introduction to Gynecology.
- (6) Schumann, E. A.—Text Book of Obstetrics.

C.A.M.S.I. NOTES

By A. J. DENNE.

It is with some trepidation that we take over the chore of pounding out the CAMSI Notes from the most capable hands of Harold Copp. He has done an excellent job and his good work has helped greatly to promote the interest of our organization.

A meeting of the local branch of the CAMSI was held shortly after the annual Medical elections at which the newly elected Vice-Presidents for the first five years of the course were present to take over their positions as local committee members. Paul McGoey was in charge of this meeting and the organization of the new executive was quickly set up and arrangements for an open meeting made.

This meeting was held on Friday, March 10th, and despite the advertising of the date, place, and hour by poster, the attendance was rather disappointing if its small size is to be taken as an indication of general student interest in an organization that can be of inestimable value to all of us. However, it is our personal conviction that a little publicity or possibly propaganda of an informative nature will stimulate wider interest and to that end the new committee is planning next year to print and distribute in some form as yet undetermined, a complete record of the organization, aims and achievements of the national and local CAMSI.

Paul McGoey and William Kerr have done a splendid job as directors of the local CAMSI and although they are still active as national executive members, the new chairman of the Toronto Local is Lloyd Hisey who has already taken hold and admirably handled the last open

meeting. Jack Denne is the new secretary.

At the organization meeting already mentioned, certain committees were set up to investigate special problems, etc., and preliminary work is now being done by these committees. At the open meeting volunteers were received to act on these which have been set up as follows:

A committee to investigate all matters related to student health, health insurance plans of various types, etc. Lloyd Hisey is chairman.

A committee under W. Caven to survey the work done and to be done in the field of Tuberculosis amongst Medical Students and interns.

A committee on Intern appointments and also Intern welfare headed by Paul McGoey.

A committee on publication to disseminate information on CAMSI activity, headed by Jack Denne, is also to cooperate with the national publications committee.

**SUBSCRIBERS ARE
RESPECTFULLY
REQUESTED TO
PATRONIZE FIRMS
ADVERTISING IN THE
JOURNAL.**



THE STORY BEHIND BLOOD TRANSFUSION

By FRANCES IRVINE, '42.

Blood transfusion is such an integral part of hospital routine that it is impossible to realize that it had to go through centuries of criticism before it was adopted as a logical procedure. Its history is a fascinating one. The ancients felt that the blood not only carried the vital force of the body, but that it was also the seat of the soul. They concluded in their groping manner that debilitating diseases and the infirmities of advancing age were due to a loss or exhaustion of this vital force while insanity was a weakness of the soul. Hence blood from healthy young bodies seemed a direct cure for these ills of wasted bodies. There was also a feeling that this might be that elusive means of perpetuating everlasting youth which was being sought after so diligently by scientists. The Egyptian papyri tell of the value of blood baths for "resuscitation and recuperation." Celsus and Pliny refer to the drinking of flowing blood from gladiators in the arena as a cure for epilepsy.

The Renaissance with its searching out of previous discoveries of the classical era, brought a revival of interest in the blood as a source of vitality. Villari's "Life of Savonarola" recounts an episode in the life of Pope Innocent VIII in 1492: "The blood of the decrepit old pontiff was passed into the veins of a youth whose blood was transferred into those of the old man. The experiment was tried three times and at the cost of the lives of the three boys, probably from air getting into their veins, but without any effect to save that of the pope." In 1593 Magnus Pegelius mentioned the possibility of transferring blood directly from the vessel of one person to those of another. Liborvius in 1615, the year before the discovery of the circulation, wrote as follows: "Let there be present a robust healthy

youth full of lively blood. Let there come one exhausted in strength, weak, scarcely breathing. Let the master of the art have silver tubes that can be adapted one to the other; then let him open an artery of the healthy one, insert a tube and secure it. Next let him incise the artery of the patient and put into it the receiving tube. Now let him adapt the two tubes to each other, and the arterial blood of the healthy one, warm and full of spirit will leap into the vessel of the sick one and immediately will bring to him the fountain of life and will drive away all langours."

The credit for actually performing the first transfusion on a human goes to Jean Denys, physician to Louis XIV. But the desire to attempt transfusions clashed with the practical disadvantages experienced in the operation. It has been said that personal feelings, politics and religion were all involved at one time or another in the waves of enthusiasm and neglect in the history of transfusion. This is evident in the decree of the French Chamber of Deputies in 1668 stating that "for the future no transfusion should be made on a human body, but by the approbation of the physicians of the Parisian Faculty." For the next century and a half only a few references, essentially derogatory, appear in the literature.

In 1824 Blundell unearthed the practice once again and after scientific investigation, placed it on a firm clinical basis. His significant conclusions were:

1. Blood loses none of its life giving properties by passing through a syringe.

2. Venous blood may be successfully used in transfusion, but arterial blood is preferable.

3. Only blood from the same species of animals is miscible.

4. Small amounts of air injected into the veins are not lethal. He recommended methods which have been elaborated to give the basis of modern technique. Associated discoveries of the 19th century tended to strengthen the revived practice. Broxton-Hicks, in 1868 treated blood with sodium phosphate to reduce coagulation during transfusion. Creite described the clumping of human red cells when they were placed in sera of cats and dogs. Between 1875-1880 the efficacy of physiological saline infusions was demonstrated, and it supplanted transfusions for a time. However, interest had

been stimulated in the older process, and investigation went on. Landsteiner in 1900 showed that human blood contained isoagglutinins capable of agglutinating other human red corpuscles, and subsequently he divided human bloods into three groups. In 1907 Jansky worked out the reciprocal agglutination reactions of some groups of blood and classified them accordingly. In the following year Otlenberg developed the clinical method of typing human blood.

At present the procedure of transfusion is used extensively with never a thought of the struggle of the so-called fanatics of past times. It is difficult to realize that only one hundred years ago the operation was looked upon as a dangerous experiment in which the mortality rate was 50%, and that even the proponents of the practice stressed that transfusion should be used only in the "most desperate cases."

CANADIAN LABORATORY SUPPLIES LIMITED

Canada's Leading Laboratory
Supply House.

Headquarters in Canada for
LABORATORY APPARATUS
AND CHEMICAL REAGENTS

TORONTO-5, ONT.
Hartz Building. 32 Grenville Street

MONTREAL, QUE.
296 St. Paul Street West

WINNIPEG, MAN.
Phoenix Building 388 Donald Street

SAINT JOHN, N.B.
108 Prince William Street

MEDICAL MANNERS



Confident Reassurance

By kind permission of Mr. Wm. Steig and
The New Yorker.

Medical Society Notes

BILL ORTVED, Secy.-Treas.

It hardly seems possible and yet sad to say the final exams are just around the corner. Since this is the case the Medical Society of 1938-39 reaches the end of its term and turns the reins over to the newly elected executive.

In scanning the activities of the past year, Cam Gray, the president, can be congratulated on the efficient handling of the Society. The Society as a whole has worked together extremely well, and each man has carried out his duties well. The various committees and the Athletic Association, under the Medical Society have turned in reports which are in the main, favorable.

The students of the faculty have certainly received full benefit of their athletic fee of \$1.50, for a special grant in excess of this, was made to the Athletic Association, to enable the purchase of much new rugby equipment and extra uniforms for the additional volley ball and basketball teams fielded this year.

The Daffydil Committee was able to prove itself again this year a self supporting enterprise and shows a profit. A portion of this profit may be utilized to start a script library and purchase certain costumes for use in future years.

The Medical Journal under John Belton was able to produce an even better and more rounded publication than ever before, and still managed to keep within the quota granted by the students of one dollar per capita.

The Medical At-Home Committee despite the fact that the dance was held on a Thursday evening this year resulting in poorer support from the students, was able to come within a few dollars of breaking even.

This year following the procedure started last year, the Graduation Banquet Committee under Bob Braiden was granted one hundred dollars to help defray expenses and lower the usual price of \$3.50 to \$2.50 per person. This showed its worth in that practically 100% of the sixth year men attended the gala affair.

The Medical Society regrets that there have not been more open meetings this year. This situation will be rectified most certainly next year, and if enough interest is shown, talks on current affairs outside of Medicine, and debates by students in the faculty may be organized.

The Society will again be able to turn

several hundred dollars over to the Bursar for use as Bursaries for Medical students. Due to the fact that a number of coats have been stolen from the Medical Building, a portion of this money may be retained in order to start a system of locks for coats in the hallways.

Medical Athletics

M. P. TOWNSEND.
Secretary-Treasurer.

Another year has drawn to a close and with it has ended the Athletic endeavours of the faculty.

Our efforts have met with varying success and as far as championships go we have done moderately well. However, if we wish to look at it from the Reed Trophy viewpoint things are as consistently mediocre as last year. This year we had twenty-one teams competing in Interfaculty sports, two more than last year and four more than the previous year. With this increase we have achieved the desired results of bringing more men into sports and have at the same time, unfortunately increased the expenses because of this to a terminal point. If we are to continue our policy of entering more teams and so bring more men into sports, which is apparently the desire of the students, we must follow one of the plans suggested in the last article.

As for a summary of season's sport there is very little to say. We have succeeded in capturing three championships this year. Namely Football, Soccer and Baseball. The Football Mulock Cup championship although perhaps the best known and famous, was not as statistically surprising as the Soccer Championship. Because this was the first time since its donation in 1894 that "Meds" had won the Arts Faculty Cup. The Spalding Cup emblematic of Baseball supremacy has returned after a two year's stay with Dents. In the previous two years our teams have been put out by Dents by narrow margins but this time the decision was reversed.

Hockey, Waterpolo and Basketball, saw our teams reach the finals but to no avail.

Again we fell behind in B.W. & F. and

Track and Field while our Swimming team made a creditable showing despite a rather small entry.

The Kinnear Trophy has again been won having run through a long drawn out course liberally sprinkled with defaults. It seems unfortunate that even the class that donated it to perpetuate the memory of one of its number should prove so uninterested. It has been questioned if such a tournament so poorly contested can serve its intended purpose and might not better be discontinued.

It has also been suggested that part

of the initiation of next year's Freshmen should be their entry in the Interfaculty Harrier contest with just rewards for those making creditable showings and just rewards for those not.

It is our opinion that although only a sprinkling of championships fell our way the true purpose of athletics has again been achieved this year. Namely the physical development of the students and the promotion of a faculty spirit.

However, this is not a philosophical lecture so may we close by saying the Executive wish you a highly successful year of sport in the next term.



Feature Section



J. C. McCULLOCH, EDITOR

THE DOCTOR IN THE CASE OF WAR

The recent European "Crises" have led many of us, who have never seen, or were too young to remember, our country in the time of war, to wonder what would have happened to us if peace had not been maintained. Where would we have gone? What would we have done? To partly outline the possible answers to these questions the author has enquired concerning the following. How were the medical services of the British Armies organized during the last war? Where did Canadian doctors go? What did they do? What medical arrangements were made in England during the last "Crisis" which ended in the "Peace of Munich"? What scheme has been laid out for Canada?

During the Great War the British Medical Corps was under the Director of Medical Services, on the staff of the General Command. Under him Deputy D.M.S.'s were in charge of the medical services of the various Army Corps. Assistant D.M.S.'s, responsible to their Deputies, commanded the medical units of the various Divisions.

The medical units in a Division can best be illustrated by tracing what happens to a man wounded at the front line. He is first taken to the Regiment-

al First Aid Post, a dugout at the trenches. There the Regimental doctor does any simple dressings. If the injury is at all serious, he is evacuated by the Field Ambulance Unit to the First Dressing Station where very minor operations and treatments are undertaken. If the man will be incapacitated for more than a few days he is transferred by motor ambulance to the Casualty Clearing Station. This is a small moveable hospital where medical men, surgeons, an X-ray unit and nursing service are present. Here the first major operative treatment of injuries is undertaken. If the patient will soon be able to return to his unit he is transferred to a Rest Station for convalescence. If recovery will be slower, he is sent to a Degassing Station, a Stationary Hospital or a Base Hospital.

Stationary and Base Hospitals are complete hospital units containing all services and capable of taking care of any case. The former contains 520 beds and can be moved; the latter 1,040 beds and is fixed. A patient is discharged from here to the Infantry Base Depot or England. The other medical men in the army were Sanitary Officers—one connected with every Division—and

directors of the different specialities.

At the beginning of the war the various medical units were sent to France, complete. Casualties among medical officers were higher than among the officers of any other similar corps in the army. Doctors for replacements were sent for 6 weeks to a training camp, where they learned to form fours, dig latrines, etc. They then went to the base hospitals, where they remained as assistants till the D.D.M.S. sent them to their final posts—such as at a First Dressing Station. Canadians, during the War, worked in all the units outlined above. When the Canadian Expeditionary Force went to France more doctors applied than there were posts to be filled. At that time the British Army was in urgent need of doctors, so many Canadians joined the Royal Army Medical Corps. Later Hospital Units, similar to the Base Hospitals mentioned above, were sent from Canada. The personnel of the No. 4 Hospital Unit, which went to Salonika, was supplied from the staff of the Toronto General Hospital. After the Salonika Campaign, in December, 1917, this Unit was transferred to Basingstoke, England, to function as a convoy hospital. And finally, a few Canadians joined the Royal Navy.

In England, to-day, doctors are associated with the fighting forces by several arrangements. Appointments in the Army and Navy are full-time. Men only become reserve doctors after several years of service. Medical officers of the Territorials, similar to our Militia, appear for a number of dress parades and spend two weeks a year in camp. They receive pay only for their days in camp. The Royal Air Force has a few full-time Medical Officers, but most airdromes have part-time doctors, from the neighbourhood, who come only for morning sick parade.

In London, during the Munich Crisis, plans were made hurriedly for medical care in case of air raids. It was estimated the first raid on London might cause 60,000 casualties. For such eventualities most of the hospitals were

to be commandeered. The staffs were to stay intact. The doctors were advised not to join the army at once, but to remain with their hospitals. These were then emptied of patients. Those who could go being sent home, the rest sent to country hospitals within a fifty-mile radius. The buildings were then sandbagged, the out-patient departments made into Dressing Stations, degassing chambers set up, and arrangements made to do the surgery and fill the wards as the casualties arrived.

Since then a more complete scheme has been made to include the whole of England and Scotland. A volunteer registration, undertaken by the British Medical Association, now has received replies from 95% of the profession. Under the Central Emergency Committee, Directors have been appointed in the various industrial districts to arrange that beds and hospital staffs will be available in case of air raids. More beds, mattresses, pillows have been bought to increase the capacity of the existing hospitals. Ambulance services are being organized and First Aid Posts placed wherever they may be needed. Medical care for children's and refugee camps in the country is being attended to. A Central Emergency Committee to organize nursing and a nursing auxiliary has been formed. The Army, Navy and Air Force will recruit Medical Officers as needed. These are only a few of the activities carried on to make England medically prepared for the eventualities of war.

Finally—What scheme has been laid out for Canada? This country is divided into Military Districts, each with its Medical Corps. Toronto, for example, is the center of such a District. Here we have the medical units for one Division—including 3 Field Ambulances, 1 Casualty Clearing Station and 1 Field Hygiene Section. Besides these there is one complete hospital unit, on paper. There are about 30 Regimental and 2 R.C.A.F. doctors in Toronto.

If an expeditionary force was organized more units would be formed and more doctors would be needed. It

has been suggested that a Secretary be appointed from the Military Section of the Canadian Medical Association to register all doctors, similar to that done by the B.M.A. With this information it would be possible to quickly place men in posts for which they are most qualified. The examination of recruits could be controlled by the Department of Pensions. This Department is continually in touch with doctors concern-

ing the physical condition of applicants for pensions. It could easily turn its activities to directing the examination of recruits.

When the laymen decide to form an army it behooves the doctors to be ready with good suggestions and concrete action. Otherwise the laymen will arrange the medical services as they want them and the doctors will march in tune, and like it.

A SUMMER-HOTEL PRACTICE IN GEORGIAN BAY.

By CHARLES SHEARD, '39.

After applying to Mr. MacDonald in the S.A.C., last spring—I obtained a position as House Physician at the Minnicog Yacht Club—Sounds very grandiose doesn't it? My duties, however, went further than mere Medicine—I was to be the leader and pianist in a three piece band and also Life-Guard four afternoons a week. With this somewhat awe-inspiring career awaiting me I set out in June for Georgian Bay. Arriving at Penetang; a small, almost-sea-worthy launch took our party (consisting of myself, my wife, the orchestra and two girls in Pass Arts going up as waitresses) through ten miles of assorted water-jumps and waves, to Minicog Island, our destination. It was typical Georgian Bay country—rocky and often windy—but nevertheless very attractive.

For the first two weeks my only patients were little girls from Detroit or Toronto who were taking a series of injections for hay-fever. Then all of a sudden I began to get a great deal of minor surgery — Whitlows, Boils, Burns, Cuts, Sprains, etc. I had good prompt results with most of my cases (hardy people these French-Canadians). That is with one exception—this case was a young maid in the Club who had sprained her ankle badly. I did it up in a pressure bandage, wrapped an ice pack around it and put her to bed. In

about an hour I was called frantically to her room—she was having terrible cramps! What on earth could be causing her cramps? I was at a loss. Her abdomen was soft, no tenderness—. It was exceedingly puzzling. A chance remark put me on the right track, however, and I discovered my patient was menstruating and that in some mysterious fashion the ice-pack was causing her cramps. Removal produced a cure and I felt somewhat ashamed of my Gynecological knowledge after that.

Another important part of my practice was the prescribing for hang-overs—and I heartily advise any who venture into the realms of resort-practice to go prepared. This particular resort, or Club, to give it its real name, was the proud possessor of a rather unique bar. The bar was constructed and decorated after the manner of the deck of a ship. It contained wheel-house, wheel, bridge, mast and signal flags, and a ship's bell. The ship's bell served to call the steward (with whom I very soon became intimately acquainted.) As you may well see it was quite an important part of my practice. I found after much experimentation that two tablespoons of milk of magnesia on retiring is the best treatment, and failing this a mixture of acetyl salicylic acid and seidlitz powders in a glass of water

in the morning—I speak with authority.

On the more discouraging side there was Poison Ivy. My treatments had no more effect on it than an expectoration on the ocean. Two of my cases had to go to Toronto and take Poison Ivy Toxoid. I've often wondered how it worked. Another case came to me, complaining of "rest-pain" in the feet—a woman of sixty. There was no pulsation in the Dorsalis Pedis and none that I could feel in the Post Tibial artery. I asked her if she had Diabetes and she replied that a doctor had told her about ten years ago that she had had Diabetes but that she had never restricted her diet much, and hadn't paid much attention to it. It took me two weeks of talking to get her to see her doctor when she went back home but in the end she promised she would. Perhaps I should have kept my mouth shut!

The most interesting case I had was a dead one. One night I was awakened by the watchman—I was wanted at the dock—in a hurry! Heart attack! As luck would have it, it was pitch black and pouring rain. The watchman's Great Dane plodded silently beside us. (I thought of the Hound of the Baskervilles.) We reached the dock and saw a harrowing sight.—In the stern of a small sloop lay my patient quite dead (a ruptured aneurism of the aorta I found out later.) His

son was quite hysterical with grief, and suffering from the cold and wet. I had to shinny down the mast, the dock was quite high, and clamber through much water to reach the dead man. By this time most of the Club employees had gathered round. Not one made a move to help—they were all French-Canadians and superstitious beyond belief. The body weighed well over two hundred pounds and whenever it was moved it croaked in a horrifying way. Well to make a long story short we finally got the body to Penetang to the morticians but it was a very upsetting night. I found out later that the sloop was becalmed at dusk—about a mile from shore—the man and his son had tried to paddle in—the exercise was too much for the old chap and his aorta popped—they lost their paddles and the son had been groping around in the dark for over an hour. Poor fellow an experience like that and on a night like that!

But to get back to more practical things. I should think that a summer spent in any one of the many summer-resorts which dot Ontario would be very broadening, and fairly profitable for any student. A word of advice though—get the hotel to charge the guests for their medical services and out of that pay you. Students of course cannot charge patients, and anyway there is something about a hotel manager.

Mallory's

"Pathological Technique"

- A practical manual written for workers in the field of Pathological Histology.
- Includes directions for performance of autopsies and for Gross and Microphotography.

434 pages, illustrated. \$5.00.

A new edition of Wolf's "Endocrinology" is promised shortly.

McAinsh & Co. Limited

Dealers in Good Books Since 1885

388 Yonge St.

Toronto

We invite you to examine these in our reading room

Abstracts

D. H. COPP, *Editor*

Contributors. { G. A. LANE
L. J. LESTER

R. L. MACMILLAN
J. C. MCCULLOCH
J. C. RATHBUN

H. J. SULLIVAN
J. G. WATT

NON-MALIGNANT STENOSIS OF THE ESOPHAGUS

By G. G. TURNER, *B.J.S. Jan. 1939.*

The writer prefaces this article with an interesting account of his personal experiences with benign strictures of the esophagus.

Causes of non-malignant stenosis

Most cases are due to the swallowing of corrosive solutions. The condition may also follow trauma. Fibrous stricture may result from a healing ulcer. There is the possibility that regurgitation of acid stomach contents with erosion of the mucosa and subsequent stricture occurs. Some cases can only be accounted for by presupposing a congenital narrowing. An inherent defect in the neuromuscular mechanism, with esophageal dilatation and stasis may allow of inflammatory changes and finally fibrous stricture.

Site

The usual sites are at the junction of pharynx and esophagus, at the crossing of the left bronchus and at the termination of the esophagus.

Symptomatology.

There is a history of gradually increasing dysphagia till finally there is extreme difficulty in even swallowing water. Finally saliva accumulates in the esophagus and is regurgitated at intervals. Due to superadded spasm the condition may be worse at some times than at others.

Diagnosis.

Although the etiological factor often cannot be found, it is usually easy to say whether the stricture is benign or malignant.

Management of Simple Stricture.

The routine plan is to dilate these strictures under the eye by means of the esophagoscope. Tapering bougies are passed in succession up to as large a size as can be got through at one sitting. Such dilatations are repeated at regular intervals. Traumatism should be avoided. The technique of self-dilatation can often be taught the patient with great advantage.

Gastrostomy is often a necessary preliminary. More rarely dilatation is of no

avail and direct surgical attack with its attendant difficulties is required.

G.A.L.

REGIONAL ILEITIS.

By DICKINSON ET AL—*Americ. Journal Surg. Mar., 1939.*

Regional ileitis is a term first coined in 1932 to describe a non-specific inflammatory disease occurring in the distal portion of the ileum.

Etiology—It is most frequent in young adults. The exact etiology and pathogenesis are unknown.

Pathology—of regional ileitis is that of a progressive, non-specific, proliferative, inflammatory process. Longitudinal ulcers appear in the mucosa. Gradually the ulcers may extend and form fistulae. General peritonitis is prevented by extensive adhesions. At the same time there is marked proliferation and repair. Eventually the involved bowel becomes stenosed, and the proximal intestine dilates and hypertrophies.

Symptoms.—Most cases have a history of years. Since the lesion is commonly in the distal ileum, the majority of symptoms are referred to R.L.Q. of abdomen. The symptoms may be divided into four groups:

(1) **Resembling acute appendicitis.** The onset is usually slower than in acute appendicitis.

(2) **Resembling Ulcerative colitis.** History of colicky pain with diarrhoea, but rarely blood.

(3) **Obstruction,** usually incomplete, may be the first symptom.

(4) **Fistula cases**—may follow exploration and drainage, or may be internal.

Diagnosis. History is suggestive. On examination there is moderate distension, and a palpable mass in over 50% of cases. X-ray is of great help. The typical lesion shows filling defect of terminal ileum, gradual tapering proximally. There is a thin linear shadow extending through the obstruction. Barium enema is usually negative.

Treatment. Appendectomy has cured some cases. If abscesses are present these must be drained. Ileocolostomy gives permanent relief in some but recurrence in

others. Resection may be necessary, and must be complete to be effective. The mortality varies with severity and duration, the average being about 12%.

L.J.L.

THE MODE OF ACTION OF DRUGS OF THE SULPHONAMIDE GROUP.

By J. McINTOSH and L. E. H. WHITBY—*Lancet*, Feb. 25, 1939.

Theories explaining the action of these drugs fall into four main groups:

(1) The drugs stimulate specific and non-specific body defences.

(2) The drugs neutralize toxic bacterial products.

(3) The drugs act on the bacteria themselves as germicides or by interfering with their power to invade and to multiply rapidly.

(4) A combination of two or more of the above explanations.

Observations.

(1) **The effect on leucocytes and phagocytes:** the administration of the drugs to rabbits and mice for four days shows no change in the level of blood leucocytes, and no stimulation of phagocytosis.

(2) **Specific antibodies and specific protection:** The immunity produced when the drug was given and living or killed cocci were injected, had the same quality, quantity and speed of production as that produced when only killed cocci were injected. The drug did not stimulate body defences.

(3) **Effect on bacterial growth in vivo:**

(a) The appearance of the organism: cocci from the blood stream show no change. Cocci injected into the peritoneal cavity show degeneration of their capsule: this effect is only obtained in vivo.

(b) The drug has no immediate effect on the multiplication and invasive power of the cocci.

(4) **Effect on the bacterial growth in vitro:** Citrated blood containing M. & B. 693 was incubated at 37 ° C with different numbers of cocci. The following observations were made:

(a) For the first few hours the bacteria multiply normally. This 'lag' is also observed in vivo.

(b) A relationship exists between the effective concentration of the drug and the number of bacteria present.

(c) The concentration of M. and B. 693 is unchanged at the end of 48 hours, that is to say, nothing is used up.

(d) The drug acts best on young virulent organisms.

Taking into account the above observations a hypothesis is formulated.

Since no fixation of the drug occurs it cannot act in the same manner as bac-

tericides, such as acroflavine and brilliant green. The latter show no lag. It is suggested that the drug acts on some essential food substance or enzyme of the bacteria. At first the invading bacteria carry over the essential factor and can multiply until the factor is used up. This produces the lag. Less virulent cocci do not multiply so rapidly and thus need less food: the carried supply lasts longer. A virus uses different foodstuffs than bacteria and so it is not surprising that this drug produces no effect on virus infections.

R.L.M.

ACCESSORY RENAL VESSELS.

Their Influence in Certain Cases of Hydronephrosis.

By HUGH JUDGE JEWETT, M.D., *Baltimore, Maryland—Surg. Gyn. and Obs.*, 1939, March, 68, 666.

The author discusses the findings at operation of thirty cases of hydronephrosis, in which accessory vessels were found at the lower pole of the kidney.

In twenty of these cases the accessory vessels appeared to cause the obstruction. The author describes the mode of formation of aberrant kidney vessels, and discusses his views on the above theories. He outlines the types of operation performed, and results, in this series.

J. C. McC.

PAUWEL'S RECLINATION.

A Physiological Reconstruction for Non-United Fracture of the Neck of the Femur.

By GEORGE J. KARFIOL, M.D., *San Francisco, California—Surg. Gyn. and Obs.*, March, 1939, 68, 648.

The author introduces into the American literature the principles of an operation designed to stimulate union of fractures of the neck of the femur.

Pauwel has shown that pressure exerted on the united head of the femur is transmitted at the fracture line as a "shearing" and as a "pressure" force. The "pressure" force stimulates the formation of bone and bony union, the shearing force stimulates the formation of fibrous tissue and fibrous union. He found that when the line of fracture was at more than 30° to the horizontal, fibrous union took place; when at less than 30° bony union. All fractures in which the fracture line is more than 30° from the horizontal are treated by subtrochanteric osteotomy, as described by Schanz. By this means the fracture line is brought to within 30° of the horizontal, and bony union takes place. Results so far obtained are excellent.

J. C. McC.

LOW BACK PAINS AND SCIATICA.

By J. A. LEO WALKER, B.A., M.D., C.M.A.J.,
Vol. 40, No. 3.

This paper is an excellent summary of the present state of knowledge concerning that difficult subject, low back pain. The author uses a somewhat modified version of the classification of Ghormley of the Mayo Clinic. This is:

1. Primary lesions of sciatic nerve-trauma, haemorrhage, or Von Recklinghausen's neurofibromata.
2. Posture. (a) Acute strain; (b) Chronic strain.
3. Trauma. (a) Involving the vertebrae; (b) Involving discs.
4. Infections. (a) Of demonstrable etiology; (b) Of unknown etiology.
5. Metabolic and senescent conditions. (a) Hypertrophic changes; (b) Osteoporosis; (c) osteo malacia.
6. Congenital anomalies. (a) Spina bifida; (b) Sacralization; (c) Spondylo-listhesis.
7. Neoplastic conditions. (a) Benign; (b) Malignant.
8. Neurological conditions. Tumours of the cord.
9. Lesions in neighbouring systems. (a) G.I.T.; (b) G.U.T.

Further description of the article is superfluous. The paper is one which should be read by every student, as the problem of low back pain is one which is becoming increasingly perplexing and important.

J.C.R.

THE ABOLITION OF CLINICAL TUBERCULOSIS BY ANTICIPATION AND CONTROL

By WILLIAM OGDEN ET AL—C.M.A.J., Vol. 40, No. 3.

This paper summarizes the results of the controlled observation of a series of 1,300 tuberculosis contacts over a period of 15 years. Each case was followed by tuberculin tests, serological tests, history and physical examination, with X-ray where indicated. It has been found that all cases of clinical tuberculosis give a preceding positive serological reaction before the onset and that 43% of those persons with a positive or suspicious serological reaction will develop clinical tuberculosis at some time after the initial observation. With this as a basis the authors by careful hygienic measures in those with positive serological graphs, reduced the incidence of tuberculosis in the controlled group from the expected 20% to 4%. In a series of 400 similarly controlled normal people no cases of tuberculosis developed while from the Framingham survey one would expect 1-2%. As a result they suggest

that all people should be examined semi-annually by the tuberculin test and all those who are tuberculin positives are then tested serologically at similar intervals. Those who eventually become serologically positive must then be placed on a most rigid routine. Tuberculosis is now added to the list of preventable communicable diseases!

J.C.R.

THE TRAUMATIC SHOULDER WITH SPECIAL REFERENCE TO RUPTURE OF THE SUPRASPINATUS TENDON

By HARRY V. SPAULDING, M.D., F.A.C.S., New York City—*American Journal of Surgery*, Volume xliii, No. 2, February, 1939.

The author reviews 1,216 cases of injuries to the shoulder joint over a five year period. He regards "bursitis" as a "refuge diagnosis," and insists all possible injuries be ruled out before making it.

Ruptures of the Supraspinatus tendon occurred in 2% of all shoulder injuries, being six times more common in males. The exciting trauma was usually indirect, due to muscle pull.

Symptoms—Usually the patient feels a sudden snap followed by severe pain and inability to abduct the arm although he can hold the arm out when it has been passively abducted.

Pathology—The tendon is usually ruptured close to the greater tuberosity and a gap from one to two and a half inches results.

Treatment—If seen early, operate. If later, when adhesions are formed consider whether or not an open operation will give satisfactory return of function. Operation is followed by immobilization in a spica with the arm at 90 degrees, abducted, and laterally rotated. The cases not operated on resulted in severe impairment of function and hence a high economic loss.

H.J.S.

BRACHIAL BIRTH PALSY

By BEVERIDGE H. MOORE, M.D., Chicago—*American Journal of Surgery*, Volume No. 2.

In this article the author reviews injuries of the Brachial Plexus due to birth injuries and describes some of the operations which may be undertaken to correct the deformities.

Etiology—This conditions arises from traction on the cords of the Brachial Plexus during difficult labours especially breech. The fifth and sixth Cervical roots are hence most liable to be damaged.

Pathology—Trauma results in perineural haemorrhages and if the blood is absorbed recovery ensues; if it cicatrizes and contracts, the pressure is permanent. If the injury is more severe it results in intra-

neural haemorrhage or even evulsion of the nerve roots from the spinal cord. The resultant paralysis is of a lower motor neurone. It may be of three types—severe, moderate or slight.

1. Severe or total.—All the muscles are affected. Flail arm in a neutral position results. The shoulder is usually subluxated. This type is rare.

2. Moderate.

(a) Upper arm type (Erb's)—arm is adducted and internally rotated at the shoulder joint; slightly flexed at wrist and fingers.

(b) Lower arm type (Klumpke's)—In this the intrinsic muscles of hand are paralyzed, and often accompanied by Horner's syndrome.

Treatment—

(a) Early—Prevent deformity and contractures by a brace holding the shoulder abducted to a right angle and laterally rotated. It is important that the brace prevents posterior slipping of the head of humerus. Massage is contra-indicated.

(b) Late—Orthopedic operations to correct deformities or transplant muscles but as the author states, the results are very disappointing.

H.J.S.

AGRANULOCYTOSIS.

CLINIC OF DR. ROBERT J. HUNTER, *Philadelphia General Hospital—Medical Clinics of North America*, Nov., 1938.

The characteristic feature of this disease is the great reduction in the total white blood cell count with few and sometimes no granulocytes. The disease comes on suddenly with symptoms not unlike grippe. There is marked muscular soreness all over the body with a high fever, often 105 F., chills coming on early in the disease. The patient feels extremely ill, looks toxic. The second most characteristic finding is ulceration. This may occur in the mucous membrane of any part of the body, but is seen especially in the mouth and pharynx.

The disease is associated with the continued use of phenacetin, cinchophen, atophan, dinitrophenol, neoarsphenamine, gold salts, quinine, sulfanilamide, pyrimidon, acetanilid, amidopyrine and many other coal tar analgesics. Individual idiosyncrasy and sensitivity are factors.

The local pharyngeal lesions of agranulocytosis can be differentiated from diphtheria, Vincent's angina, apthous stomatitis by smear, culture, and most important blood count. Aleukemic leukemia presents a picture at times that is impossible to differentiate from agranulocytosis.

Treatment.—Amidopyrine or any other drug that is thought to be a cause should

be withdrawn at once. Pentnucleotide, 10 c.c. intramuscularly, four times a day, has been used. Leukocytic cream may be used instead of pentnucleotide. Adenine sulfate, 20 c.c. daily, intravenously; nucleic acid and glutathione have been tried with some success. Blood transfusion should be given in all cases.

J.G.W.

THE ETIOLOGY AND PATHOGENESIS OF ALCOHOLIC CIRRHOSIS OF THE LIVER.

By CHARLES L. CONNOR, M.D., *San Francisco*
—*Jour. A.M.A.*, Feb. 4, 1939.

In order to orient the subject of alcoholic cirrhosis, and in order to understand the difference between it and other cirrhosis, a brief summary of the various types encountered is given.

(1) **Toxic cirrhosis** is the cirrhosis following necrotizing lesions of the liver. Among the causes are cinchophen, arsenic compounds, acute catarrhal jaundice, infectious jaundice, severe thyrotoxicosis, Weil's disease. It occurs in children, men or women indiscriminately of the use of alcohol.

(2) **Biliary cirrhosis** is always associated with biliary obstruction, with or without infection. The lesions begin in the portal areas.

(3) **Pigment cirrhosis** is a type of portal cirrhosis. It is often associated with hemochromatosis, sometimes with pernicious anemia.

(4) **Fatty cirrhosis** is a third type of portal cirrhosis in which the lesions develop around lobules, joining the portal areas into continuous bands of fibrous tissue.

Alcoholic poisoning and severe chronic alcoholism means the constant consumption of large amounts so frequently that the normal carbohydrate-fat metabolism is materially altered, or that alcohol is taken so frequently that normal metabolism may not be re-established in the intervals. This often means the drinking of two quarts a day of beer, or a quart of sherry wine (22 per cent. alcohol) per day, women preferring the wine.

Alcohol interferes with carbohydrate metabolism and fat oxidation because of its action as a cell and tissue toxin, and because during a "bout" there is starvation or lack of carbohydrate in the diet. The anoxia results in hyaline degeneration or necrosis of the liver cells and proliferation of fibrous tissue.

Types:

(1) The acute fatty liver of alcoholism develops after prolonged use of alcohol in

large amounts during which little or no food is taken.

(2) The fatty liver in which there is early, progressive perilobular fibrosis is the product of alcoholism that is less in degree. The liver is large, or normal, and smooth.

(3) The third phase liver is small in size, but not as small as the liver of toxic cirrhosis. This is the classic cirrhosis of the portal type (Laennec's cirrhosis). The cause is not known.

It is doubtful if a person on an adequate diet of protein, fat and carbohydrate plus alcohol will develop cirrhosis. A person who eats three good meals a day will not likely become a chronic alcohol addict. It is possible to maintain metabolic equilibrium within safe limits even when large amounts of alcohol are taken habitually, if sufficient food is taken.

J.G.W.

Book Review

"I SWEAR BY APOLLO. A LIFE OF MEDICAL ADVENTURE"

By WILLIAM E. AUGHINBAUGH, M.D., *Farrar and Rinehart*, 1938.

A chance remark appearing about half way through this book sums up very completely the character of its author. Dr. Aughinbaugh at one point in his career set out from his native United States for Peru, in order to study verruga perviana, variously known as canyon fever and Carrion fever. When Dr. Aughinbaugh reached Lima, he found to his pleasure, and at the same time to his disappointment, that a scientific expedition from an American college had preceded him and had demonstrated that the disease was due to the bite of a night flying gnat. Much of the danger had already been eliminated by the provision of mosquito netting and skilled laboratory work had devised a serum as well as a technique for treating victims.

"Only the details of application and organization remained to be done, work in which I could not be of great use; nor was I particularly interested in this phase of medicine."

"Nor was I particularly interested in this phase of medicine." Indeed, Dr. Aughinbaugh was not interested in this phase of life. One cannot but stand amazed at the way he hops from country to country, job to job. Not application and organization of medicine, but discovery and adventure lured him on, until the story he tells becomes almost unreal to most

of us. At eleven he ran away for a month's trip as "cabin boy" aboard a schooner in Florida; during the Spanish-American war, having been refused by the army because of a weak heart, he got a position as ship's surgeon on a vessel plying between New York and Cuba, chartered to carry sick and wounded soldiers home; for three years he was in charge of a leper hospital in Caracas, Venezuela; he was on the first relief ship after the eruption of Mt. Pelee in 1902, when 30,000 lives were lost; he went to India to study plague and the Koran; he travelled as medico aboard a pilgrim ship crossing the Arabian Sea to Jidda, hoping to go in disguise to Mecca — and saw Asiatic cholera break out on board, wiping out some five hundred of twenty-eight hundred passengers; on his way home from India he visited Egypt and studied trachoma, to say nothing of dropping in on most of the countries in Europe; unable to stay in New York more than a few months after he got home, he was off to the Gaspé peninsula; he helped to run a hospital in Mexico, in spite of revolution and counter-revolution; and so on — and on — and on. One cannot help admiring his wife.

This is, however, not a commercial blurb for the book jacket. Nor shall we repeat any of the remarkably good stories with which the book abounds. Most of us are bound by our profession, be it medicine, law or whatnot. The business of living is subordinate to it. Having passed our exams in medicine, we settle down to practice it. The height of our experience is, often enough, bringing babies into the world in taxicabs. Not so with Dr. Aughinbaugh. His book is given the subtitle "A Life of Medical Adventure," but the medical part of it is distinctly incidental. He is a man who loves adventurous living, whether it has anything to do with medicine or not. In one place he says, "On the medical side, too, there were little adventures." For too many of us medicine becomes a religion, filling and fulfilling life by itself, and if we ever thought there was anything else, we forget it.

It is a great pity that this is often held up to us as an ideal. Medicine, it is intimated, should be the be-all and end-all of life. We are urged to make our lives worthwhile and successful (not necessarily monetarily) by devoting ourselves entirely to the medical ideal. Life is not such a serious thing as that, in spite of centuries of men trying to make it so. Life may be an adventure, or it may not, just as we please. For Dr. Aughinbaugh it has been an adventure in which medicine played an interesting part.

J.A.R.

CONTENTS

Volume XVI — 1938 - 39

A

- Abstracts. 1:43, 2:102, 3:159, 4:214, 5:272, 6:318.
 Actinomycosis. E. C. Evans, '39. 6:301.
 Acute Anaphylactic Shock. H. H. Varty, '41. 4:208.
 Adrenal Cortex. D. H. Copp, B.A., '39, and N. Littner, B.A., '40. 3:127.
 An Early Case History and Autopsy Report. H. Bolley, '41. 4:205.
 Appendicitis. F. G. Kergin, B.A., M.D., F.R.C.S. (Eng.). 4:179.
 A Summer Among the Indians of British Columbia. Bill Service, 3T9. 3:156.
 A Summer Hotel Practice. C. Sheard, '39. 6:316.

B

- Benzedrine Sulphate. J. K. W. Ferguson. 5:234.
 Birth Palsies. Crawford B. Shier, '40. 2:84.
 Book Reviews. 1:47, 2:108, 3:163, 4:220, 5:271, 6:322.

C

- C.A.M.S.I. Notes. 2:83, 3:155, 5:265, 6:310.
 Cerebro-Spinal Fluid Mirror. James G. Watt, 4T0. 3:137.
 Cholera Epidemic in China. J. R. Lind, M.D. 5:262.
 Corrigan Pulse. 3:158.

D

- Diverticulitis. B. Laski, '39. 3:133.
 Dysmenorrhoea. Evelyn K. Johnston, B.A., 40. 3:144.

E

- Endometriosis. Barbara Watts, '39. 4:191.
 Essential Hypertension. R. F. Gerrard, '39. 6:295.

F

- Famous Medical Schools:—
 British Post-Graduate Medical School. Kent Harrison, M.D. 3:120.
 Cornell University Medical College. LeRoy O. Travis, A.B., '39. 2:79.
 Harvard Medical School. Lawrence J. Stuppy. 1:18.
 Johns Hopkins Hospital and Medical School. F. T. Larochelle. 5:236.
 Saint Bartholomew's Hospital, E.C. Christopher Evill, M.B. 6:293.

- First Aid on the Highways. William Butt, '40. 1:34.
 F.R.C.P. & S. Regulations. 5:241.
 Fundamental Principles of Nutrition and Their Practical Application. Frederick F. Tisdall, M.D., F.R.C.P. (C). 3:113.

H

- Heparin. L. B. Jaques, M.A. 4:185.
 Hippocrates. R. M. Archibald, '39. 4:212.
 Hypoparathyroidism and Tetany. R. F. Farquharson, M.B., F.R.C.P. (C.), 1:4.
 Hypoparathyroidism and Tetany; Part II. R. F. Farquharson, M.B., F.R.C.P. (C.) 2:53.

I

- Indications and Uses of Sedatives in Medical Practice. A. Auerback, M.D. 6:283.
 Infections in Hands. J. Harold Couch, M.A., F.R.C.S. 5:225.

M

- Medical Athletics. 1:41, 2:101, 5:270, 6:313.
 Medical Interlude. E. P. Scarlett, B.A., M.B., F.R.C.P. (C.). 4:167.
 Medical Opportunity in China. R. B. McClure, M.D., F.R.C.S. (Edin.). 2:98.
 Medical Society Notes. 1:40, 2:101, 4:213, 5:269, 6:313.
 Microscopic Examination of Urine. Vera J. Binnington, '39. 1:22.
 Modern Treatment of Pneumonia. W. Hurst Brown, M.A., M.B. (Oxon.). M.R.C.P. (Lond.). 2:65.

O

- Organized Medicine in Ontario. A. D. Kelly, M.B. 2:59.

P

- Physiology of Essential Hypertension. T. S. Wilson, '41. 3:152.
 Problem of a Lump in the Breast. W. A. Rowland, '40. 3:152.
 Pruritus Ani—Its Classification and Treatment. James T. Danis, M.D., F.R.C.S., (Edin.). 6:280.

R

- Rediscovery of Observation. A. F. Coventry, B.A. (Oxon.). 1:10.
 Remarks on Chemotherapy. P. H. Greey, M.D. 5:229.

Reticulo-Endothelial System in Relation to Hodgkin's Disease. R. A. G. Lane, '39. 5:253.

S

Scope of Plastic Surgery. Stuart Gordon, M.D., F.R.C.S. (Can.). 3:118.

Some Principles of Value in the Diagnosis of Abdominal Disease. Hubert Hayward, B.A., M.D. 2:75.

Sternal Puncture and Bone Marrow. Paul Greenberg, B.A., '40. 2:96.

Story Behind Blood Transfusion. Frances Irvine, '42. 6:311.

T

Tests of Liver Function—T. A. Fraser, B.A. '40. 5:257.

The Doctor in Case of War. 6:314.

Thyroid Gland. D. J. Breithaupt, '41. 4:202.

Travel by Cattle Boat. A. Lane, '40. 4:211.

Treatment in Congestive Cardiac Failure. C. W. Taylor, '40. 2:92.

Treatment of Diabetes Mellitus. M. P. Hoover, '40. 4:197.

Treatment of Gonorrhoea. Noble Black, M.B. 4:172.

Tubal Pregnancy — Pathogenesis and Symptomatology. A. M. Sutton, '39. 6:304.

V

Venous Thrombosis and Pulmonary Embolism. C. C. Gray, '39. 6:287

Vitamin A. Wallace W. Graham, M.D., D.D.S., M.R.C.P. 1:13.

Vitamin B. Wallace Graham, M.D., D.D.S., M.R.C.P. (Lond.). 2:70.

Vitamin B₂ Complex. Wallace Graham, M.D., D.D.S., M.R.C.P. 3:122.

W

Water Balance and Its Relation to Surgical Conditions, Part I. G. A. Lane, '40. 5:242.

Water Balance and Its Relation to Surgery, Part II.—L. J. Lester, B.A., '39. 5:248.

Why Hospitalization for the Mentally Ill? A. G. Shulman, '39. 1:29.

THEY ARE HELPING US—HELP THEM

The following is a list of those whose ads. appear in this issue. If you need their products or services, we recommend them to our readers for patronage:

AYERST, McKENNA & HARRISON, LIMITED	27
CANADIAN LABORATORY SUPPLIES, LIMITED	312
FROSST, CHARLES E., & CO.	Inside Back Cover
HARTZ, THE J. F. CO., LTD.	300
McAINSH & CO., LIMITED	317
MEAD JOHNSON & CO. OF CANADA	Outside Back Cover
WINTHROP CHEMICAL COMPANY, INC.	Inside Front Cover
WYETH, JOHN & BROTHER	278

